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DOURINE.

By CAPT. F. S. W. BALDREY, F.R.C.V.S., F.R.G.S., Civil
Veterinary Department, India.

Nomenclature.—This disease has been known by various names for a very considerable time; these are: (*a*) Common venereal disease of the horse, (*b*) Horse syphilis, (*c*) Maladie du coït, (*d*) Genital glanders, (*e*) Breeding paralysis, (*f*) Covering disease, and (*g*) Dournine. The last is, from what I can learn, derived from the Arabic, and signifies dirt (*vide* Pease's Note on Dourine); being short and distinctive, it is the preferable one, and is now generally used.

History.—The disease appears to have been known in Algeria from very early times, but was only first noticed in Europe in the beginning of the nineteenth century, and then only in Southern Europe and Southern Russia, to which latter place it was probably imported from Syria. It is now recognised in Northern Africa, along the whole of the Mediterranean Littoral of Europe, in Northern Spain, the Pyrennees, and Southern France. It is known in Persia and Asia Minor. In the United States it occurred in the De Wet county of Illinois in 1889, in a case said to have been imported from France, which, however, Nocard says is not possible. A similar disease has been described in Chili, but this has not been authenticated (Annett, *Liverpool Lectures*).

In 1840 laws embodying protective measures were passed in Poland and Silesia. It was very prevalent about the middle of the last century in Germany and its tributary states, but was suppressed by the German laws in connection with epizootics. It has never been known in England or Belgium (Friedberger,. It was at one time thought to be identical with human syphilis, and was believed to be conveyed by men to asses and mares, but Haussman and Knauert demonstrated the fallacy of this.

It was not until 1896 that Rouget discovered a trypanosome in connection with this disease, but his work was not really credited or substantiated until 1899. Then Schneider and Buffard indubitably demonstrated, by means of infecting dogs, that the trypanosome, isolated from an infected stallion, was the cause of the disease. Marek demonstrated some histological changes in nerves and their centres, and Nocard verified the experiments of Schneider and Buffard.

The disease was reported by the French Army Veterinary Surgeon Signol in Algeria, in the province of Constantine, in 1847. He described it as an Epizootic Paraplegia occurring amongst the horses of the Rigas tribe of Arabs. These people said they had known it for a very long time, and it occurred in a serious form every fifteen to twenty years. It occasioned great loss at that time. From the symptoms described by Signol there is no doubt as to the nature of the disease.

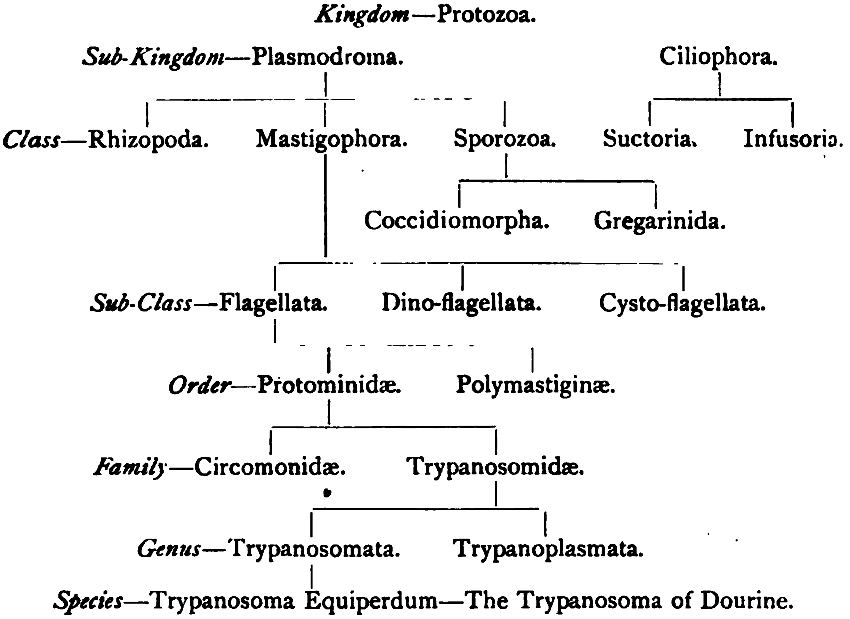
It has in all probability been existent in India for a considerable length of time, but it was not recognised as trypanosomiasis until two years ago, when Pease first demonstrated the parasite in the Punjab. I have seen what I am sure was the disease in the Bombay Presidency, when on horse breeding duty in 1899, but did not realise its significance.

Cases amongst Government stallions were recorded as suffering from ulcerated sheath or penis, sprained penis, chronic enlargement of the genital organs, emaciation, asthenia, with subsequent paralysis and death, or destruction. I again saw the disease when on duty in Beluchistan and in the united provinces in the Babugarth Government stud in India. It was during the time I was at Beluchistan in 1902 that Pease first demonstrated the presence of the trypanosome in a horse that had been at stud work in the Punjab. Cases previous to this had been recognised as having something out of the common, as the above mentioned variety of alleged causes will illustrate, and they were isolated and treated as though suffering from a specific affection, but the real cause was not known.

Etiology.—The cause of the disease is a trypanosoma, named the trypanosoma equiperdum, belonging to the genus trypanosomata.

The trypanosomata are unicellular organisms, usually provided with a flagellum at the anterior end, but occasionally in some species with one at each end. They are generally blood parasites, but may occur in the other body fluids, mucus discharges, glandular excretions, and ulcerations. The following table shows their position in the animal kingdom according to Ray Lankester's classification :—

INVERTEBRATA.



The trypanosoma is 18 to 26 microns in length and 2 to 2·5 microns in breadth. It is therefore smaller than the surra organism, which it otherwise closely resembles. It is much smaller than the trypanosoma Lewisi, and has not the chromatin granulations of that parasite. It has a well-marked nucleus and centrosome, from which latter arises a vigorous undulating membrane and flagellum, these being quite distinctly marked when examined in freshly drawn blood; it is not so active as the surra organism, and never so numerous in the field of the microscope. Multiplication takes place by longitudinal division only, as far as is at present known. Buffard described rosette forms of multiplication, but this has not been verified, and it was probably agglutination which he saw. Fresh specimens will remain alive and active for some days on a moist slide at room temperature, and will live for seven days on ice (Annett). The posterior extremity is more pronouncedly beak-shaped than in t. Lewisi; it is not so sharp as in surra. The centrosome also tends to be elongated, and is larger than in either of the above. The nucleus is large and somewhat loosely granular.

Infectivity.—Equine animals are very susceptible to natural infection, but less so to artificial infection, the success of which depends largely upon the amount of infecting material introduced. If only a small dose, the phagocytic action of the leucocytes seems to be capable of dealing with the parasite and preventing general infection (Pease), or the agglutinin normally produced by the blood of the body under the stimulation of a foreign substance has sufficient power to cause agglutination and death of the parasite. The latter I think to be more probably the correct explanation, as I have never seen any sign of phagocytosis when observing freshly drawn infected blood.

Natural infection in almost all cases is conveyed during coitus from the stallion to the mare, or *vice versa.* It is the only mammalian trypanosome which, as far as is at present known, is not conveyed by the medium of a biting insect. The parasite is found in the semen and urethra of the male, and in the vaginal mucus of the female, where it appears capable of living indefinitely. The dog is susceptible, especially young animals, and Pease has demonstrated its infection from dog to bitch by coitus; he also showed that pariah dogs in India are practically immune. In the laboratory at Liverpool the disease is maintained in dogs.

Other methods of infection may occur, but are not very probable, such as by means of litter, etc. It may, however, be conveyed through the medium of sponges or towels, which, when used to clean the vulva of the female, may afterwards be used on healthy animals (Nocard).

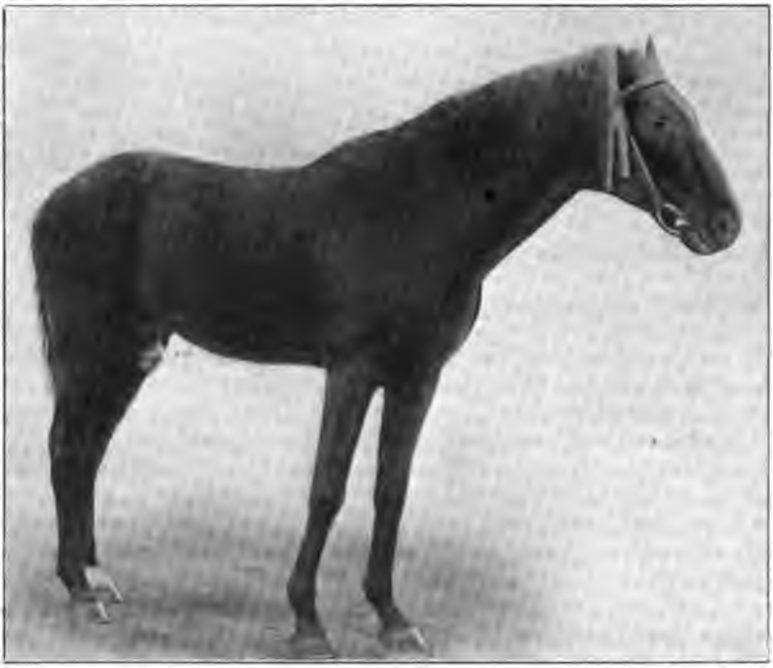


FIG. 1.

A thoroughbred English horse in the primary stage, some weeks after the onset of the first symptoms. Only enlargement of the penis is yet evident. No plaques or œdemata have yet appeared, and there has been no loss of condition. Naturally infected.

Rabbits, rats, and mice are easily infected by inoculations of blood, and may carry on the disease by coitus. The results of artificial infection by subcutaneous inoculation are similar to those of natural infection by coitus.

The ass is susceptible, but in a lesser degree than the horse, and the disease assumes a more chronic and less acute form.

Cattle have been considered immune except to an overpowering dose of infective blood, and even then the parasites do not remain for any length of time in the blood. Pease has shown this by some experiments reported in the *Veterinary Journal* for November of last year. His experiments, however, prove that a difference between dourine and surra does exist, as cattle after showing complete immunity to dourine still give a reaction to surra, to which disease they are susceptible by inoculation.

Incubation.—The incubatory period from artificial infection is from seven to ten days. By coitus the first symptoms will be seen in from twelve to twenty days, according to the amount of infective material introduced and the susceptibility of the animal.

Seven days after inoculation there will be a hot and painful swelling at the seat of inoculation. If by coitus, after twelve to twenty days, there will be a discharge from the vulva of the female, or in the male there will be urethral irritation and discharge. Pease's experiments in India place the period of incubation from natural infection at from fifteen to twenty days.

Williams, in his report on the outbreak in Illinois, says, "its commencement is far more insidious in stallions than in mares, being in many cases wholly unrecognisable for months." This is not in accord with my experience. If careful watch be kept, the initial symptoms will be seen, and more specific symptoms be palpable, in less than the period mentioned by Williams.

Symptoms.—These may be divided into three stages, as suggested by Nocard. These stages are distinct, and may, if the case is carefully watched, be recognised comparatively easily. They are:—

Primary.—In which occur the local manifestations of discharge and urethral irritation, and ulceration of the penis and sheath.

Secondary.—In which the exanthematous eruptions appear in the skin—the so-called "plaques."

Tertiary.—Characterised by the formation of lesions in the central nervous system, and by nervous disturbance with ultimate paraplegia.

It was probably on account of these three periods that the older writers confounded the disease with syphilis, and it is also possible that the ulcerations and chronic enlargements of the sheath and penis gave rise to the idea that it was a form of localised glanders.

Primary.—The first noticeable symptom in either natural infection by coitus or infection by subcutaneous inoculation is a discharge from the vulva of the female or the urethra of the male. The mucous membrane becomes reddened and swollen, and often in the male there is erosion to a slight extent. These symptoms may be so slight as not to be noticeable by the layman, unless specially warned and there is reason to suspect the prevalence of the disease. Its onset is most insidious, and for that reason the more dangerous. A stallion used for stud purposes will at this time be dangerous, and may infect many of the mares which he serves. On account of the irritation present, the horse will have frequent erections, and will be more than usually eager to cover mares in the beginning of the disease. Mares will show irritation by frequent micturition, simulating œstrum at short intervals. It has been noticed in India that from this time, although the stallion is capable of efficiently covering the mare, he is entirely sterile. The trypanosomes may be found in the semen, which possibly accounts for the phenomenon. These symptoms are concurrent with œdema of the penis, and often associated with phymosis and paraphymosis, swelling of the sheath and scrotum, and œdema along the abdomen, often extending to the sternum, and which may be of an inflammatory nature or cold and passive. These swellings have a tendency to persist throughout the disease, but they may be metastatic. Œdema often attacks the fore and hind legs. The testicles are œdematous and painful on pressure. The penis and scrotum may be ulcerated and the penis may show patches of erosion, but these are neither constant nor essential (Nocard). The ulcerations yield to simple antiseptic dressings and heal up, but they tend to recur.

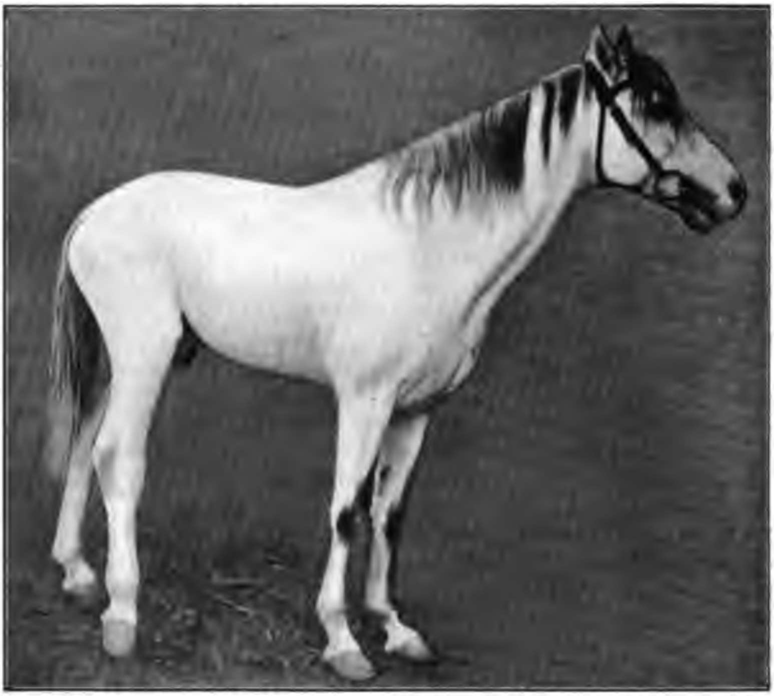


FIG. 2.

A grey Arab horse. A somewhat more acute case than the preceding, at the end of the first stage. No plaques have yet appeared, but œdema of the testicles is noticeable. There is also inflammatory swelling of the off hock joint. Naturally infected.

The temperature at this time may be slightly raised, but the patient feeds well, sometimes voraciously, and shows no constitutional disturbance. In very severe and acute forms in this initial stage micturition is occasionally painful and coitus may be impossible, but I have not myself seen this very acute form. Nocard says that the kidneys are painful, that the back is arched, and that the animal is quickly breathless after a sharp trot. These symptoms I have not particularly noticed in the early stages of the more or less chronic forms with which I am acquainted. Weakness of the loins is, however, occasionally noticeable. Although the appetite remains good the animal may begin to lose condition, which, however, is not very noticeable yet. In the female there is often intense pruritis, but this is seldom noticed in the male. The mare will also still be showing symptoms of œstrum, and, as Nocard says, "la joument prend les allures des bêtes nymphomanes." The mucous membrane of the vagina and vulva is swollen and red. These acute symptoms in the female subside after the course of some three or four weeks. The œdema becomes more general, extends along the inferior surface of the abdomen, and may affect the mammary glands. It is, however, passive and not painful, and may extend down the inside of the thighs. As in the male, if it exists, the effect of the pruritis will be seen, and as it dies off there will be in all probability some white patches of skin left, denuded of hair.

Secondary.—Cutaneous and glandular period.—After a period of four to six weeks the initial local symptoms may be said to become more generalised and distributed. Emaciation has become distinctly noticeable, and there is a certain amount of langour in the animal's movement. The loins are tender under pressure, and the ears may hang in a somewhat pendulous position. In acute cases there is some loss of co-ordination in the movement of the hind legs. Nocard mentions that the "faux pas" is now seen at the trot.

The swellings of the sheath, penis, and abdomen have become chronic from infiltration, and are hard and painless. It is at the beginning of this stage that the attendant usually first notices that something is seriously wrong with the horse, unless he has had some previous knowledge of the disease. It is also at this time that the first really pathognomonic clinical symptom appears (Pease). I speak of the so-called "plaques." Buffard says that they are first noticed on the fortieth to forty-fifth day after infection, and are distinctly characteristic. These plaques are cutaneous eruptions occurring spasmodically and irregularly at various centres upon the body, usually about the neck, shoulders, and fore part of the chest and back, but they are also seen on the loins and quarters.

They are more or less rounded and of various sizes; Nocard says from a two-franc piece to the size of one's hand. This is in agreement with what I have seen. Pease describes them as "salient and rounded and look as though they had been caused by slipping a disc of metal under the skin." I cannot think of a more graphic description. They are occasionally so slight as to be difficult to detect, and I have discovered them by looking sideways along the animal and noticing the slight horripilation of the hair over the affected spot, which effect is always produced by the erector pili muscles as a result of the irritation in these circumscribed areas. They can often only be seen by so standing at an angle to the direct light. They may be felt by passing the hand carefully over the body, as a slight thickening, but they are not at all hot or painful. They may exude a slight serosity which tends to agglutinate the hair (Nocard). The patches are very irregular in their appearance, and they may last only a few hours or up to several days, and disappear without leaving a trace. Or they may become œdematous and persist for a slightly longer period. The parasite (trypanosome) is always found most easily by puncturing with a needle in the centre of the plaque, and taking a drop of blood for immediate microscopical examination. It is this which constitutes a positive diagnosis of the disease. The 2-3rd inch objective and No. 4 eye-piece is sufficient magnification with which to observe the living and rapidly-moving organisms in the blood; the condenser should be racked well down and the diaphragm partially closed to limit the light and increase the definition. With a ⅛-inch objective and more light they can be observed quite distinctly.

Occasionally the plaques are preceded some twenty-four to forty-eight hours by an evanescent erruption of papules, which disappear in the course of the day, leaving no trace or only a slight serous exudation (Nocard).

Progressive anæmia is now very marked, and lymphatic glandular complications are noticeable. The glands become enlarged, especially the inguinal, which may suppurate (Pease). They are, however, not painful, and are at first soft, but tend to become harder from the infiltration. The appetite still remains good.

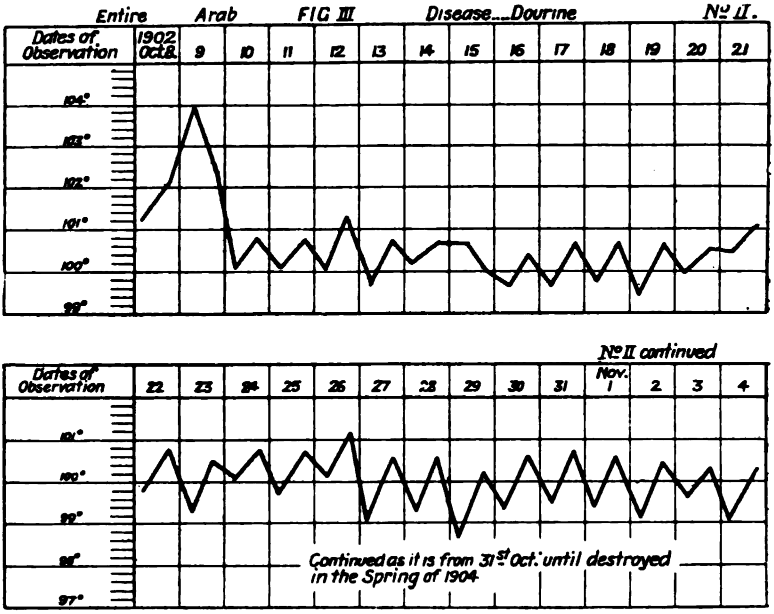


CHART 1.

Showing the variations in the temperature of the pony depicted in Fig. 3.

The animal rests a good deal in a lying posture, and occasionally experiences difficulty and pain in getting up. It is indifferent to surrounding objects, and stands listlessly in the stall.

Progression is noticeably altered, and the animal sways, especially at the trot, which is induced with difficulty. Coitus is almost impossible in the stallion, and erections are feeble and incomplete.

In mares the symptoms are somewhat similar; the discharge from the vagina and vulva persists for months or even for a year or more. It becomes viscid and irritating to the adjacent parts.

Infected mares do not as a rule conceive, but if they do they almost invariably abort at about six months (Pease).

The period at which plaques appear is the same as in the stallion.

Tertiary or Nervous Stage.—General progressive anæmia is now very rapid. The appetite becomes capricious and the temperature slightly raised. Co-ordinated locomotion is impossible, and the animal is in fact hardly able to stand without support. The symptoms of paraplegia supervene.

The mucous membranes are of a pale rose colour, the urine is thick and passed with difficulty. The animal becomes worse and worse, with indolent abscesses on the sheath and scrotum. It eventually dies if not mercifully killed. Death may be more or less sudden during this paralytic stage, and is due to syncope.

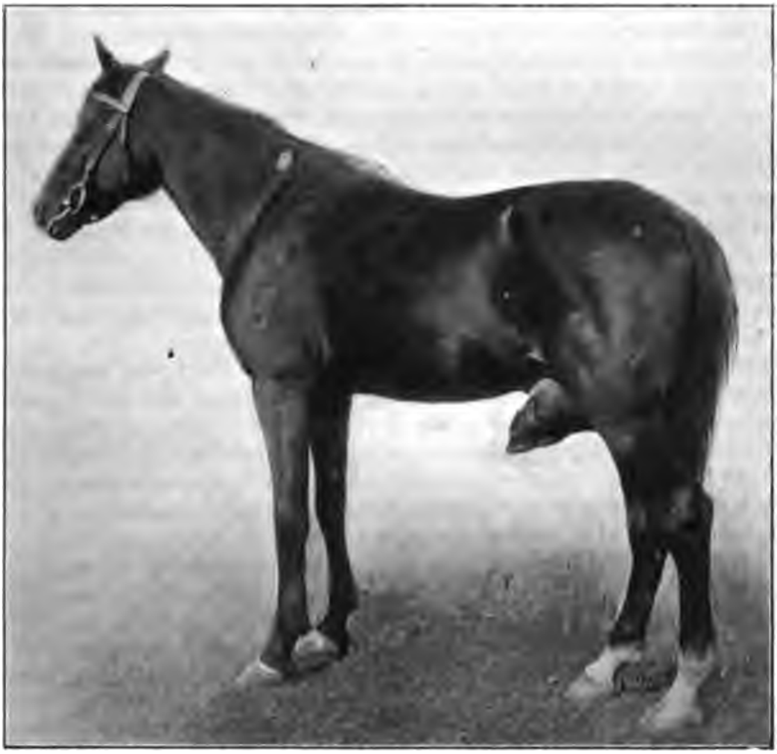


FIG. 3.

A bay Arab pony, at the beginning of the second stage. The sheath and penis are enormously swollen, cold and passive. No other œdema was noticeable at this stage except at the umbilicus, which is not visible in the photograph. Plaques had appeared, but they are not distinctly brought out in this figure. The depressed aspect of the animal is well shown. Naturally infected.

In mares the tertiary period is similar, but de Blaise says that inability to walk is not so pronounced as in horses.

The total duration of the disease is from twelve to eighteen months. Nocard says one to two years, and Buffard two to ten months. I think the first is nearest to the mark, and the latter I have not seen.

The symptoms in the donkey are very similar, but of a more chronic nature, and therefore more difficult to recognise in the early stages. The Arabs say that he may recover and become immune, in which case, for stud purposes, he is much more valuable.

Pathology and Lesions.—There are in the early stages only slight pathological changes, viz., the ulcerated local lesions of the sheath and penis, with local œdema. The trypanosome is in the general circulation, and continually reproducing its cycles of existence. At each cycle it uses up more pabulum from the body of its host, as it is a true parasite, and causes at each fresh crop a period of congestion. When examined in a fresh blood preparation, it will be seen to move actively about, pushing the red corpuscles violently to one side; but it does not appear to in any way attack the corpuscles. Its destructive effect on the corpuscles is produced by the enormous amount of oxygen which it uses up, depriving the blood elements of their normal share, and so causing corpuscular disintegration by the breaking down of the contained hæmoglobin, with consequent anæmia and cachexia. The toxines liberated by the parasite have also a very prominent share in the destruction of the blood elements. The parasites exist in the spinal fluid, and they could no doubt be found here when not demonstrable in the peripheral circulation, as is the case in the trypanosomiasis due to the trypanosoma gambiense (human sleeping sickness). This is a valuable means of diagnosis, and it might be made so in the horse (Christy, *Liverpool Lectures*).

The parasite is also found in the seminal fluid, the vaginal discharges of the female, the urethral discharges of the male, and the fluid extracted from œdematous swellings.

Inoculation, either subcutaneously, intraperitoneally, intravenously, or into the conjunctival mucous membrane, with any of these materials or blood, will produce the disease in susceptible animals. The trypanosome may be passed from horse to dog and back again, and still produce the identical disease, thus establishing its specificity. The parasite cannot be grown, so far, on artificial media, in vitro, so that it is impossible to absolutely fulfil Koch's postulates; but it is otherwise undoubtedly proved that the parasite is the cause of the disease. No matter where the inoculation is made the subsequent symptoms are the same.

Pease says in connection with the changes in the blood that there is leucocytosis, and that the eosinophile leucocytes are largely increased. Undoubtedly the former is the case, as it is in the majority of infective diseases. The fact that the horse's blood normally contains a plentiful percentage of eosinophiles, and that there has been no systematic research into the comparative numbers of eosinophile leucocytes in the horse, render it difficult to accept the alleged large number of eosinophiles in the blood as a reliable help in the diagnosis of this disease.

With regard to blood diagnosis there remains a good deal that might be done. Agglutination is easily brought about in other trypanosomiases, and no doubt could be used in the case of dourine as an aid to diagnosis. The hæmatozoon, for practical laboratory work, can always be kept in dogs, rabbits, rats, and mice, and experiments are wanted to test whether the serum from an animal which has the disease, but in which the parasites are difficult to demonstrate, would, if mixed with certain dilutions, cause agglutination of the parasites when taken from the peripheral circulation of a dog or other laboratory animal. There is a difficulty in doing this, as the protozoon cannot be cultivated in vitro, and when mixed with the corpuscular elements of the blood it is not so satisfactory to work with as an organism as when it is in an artificial pure culture. The red corpuscles may be separated by the hæmatocrit, and the parasite comes down in the layer with the white corpuscles, but it is doubtful whether the centrifugalising would affect the organisms or not, and hasten their agglutination. The phenomenon of agglutination is so pronounced in some bacterial diseases that it is feasible to hope that results of a satisfactory nature could be obtained here, and these would be invaluable, in view of the urgent necessity of an early diagnosis.

Post-mortem.—This reveals a round-celled infiltration of the sweat glands and a yellow gelatinous exudation beneath the skin, especially in the abdominal region. Fat has almost entirely disappeared from the body. The bones are fragile and may be infiltrated with gelatinous matter. The bone marrow is reddish-brown in colour. The saliva is blood-stained. Articular surfaces show erosion, which accounts for the pain which is experienced in rising in the latter stages (Nocard). There is fæcal stasis. The spleen is always enlarged, the kidneys passively congested. The myocardium is soft and pale brown. The lymphatic glands are soft, enlarged, and contain a yellowish blood-stained fluid, with small greyish foci and local hæmorrhage.

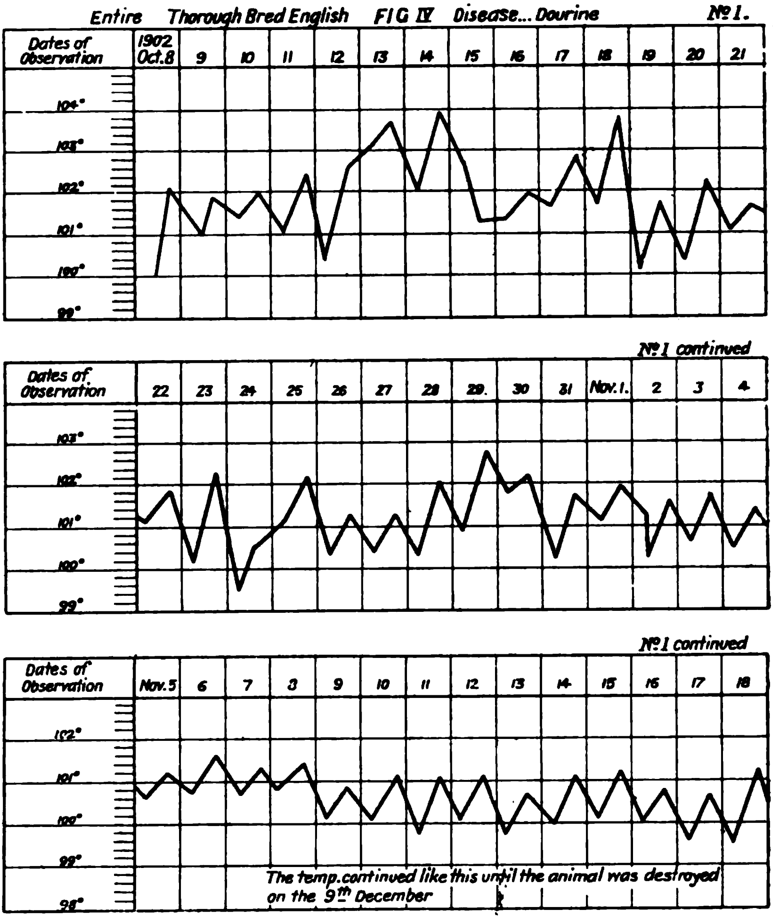
The testicles are soft and small. In many cases they have been entirely replaced by fibrous tissue. The female vaginal mucous membrane is marbled, thickened, and has hæmorrhagic patches. There are distinct organic lesions of the central nervous system. Dura pater, pia mater, and brain are dropsical. The cervical arachnoid membrane is rosy and contains an albuminous fluid.

In the lumbar and sacral regions the cord is reddish and forms a diffused pulp in chronic cases, but not in acute. Histologically the cord, but not the brain, shows degeneration of the posterior column, well marked in the cervical and lumbar regions. The grey matter is not enlarged. Nerve cells show destruction of nuclei (chromatotexis).

There is fatty degeneration and atrophy of the muscular tissue of the body, this tissue being reduced by at least one half (Annett, *Liverpool Lectures*).

Diagnosis.—The diagnosis of this disease is extremely difficult in the early stages, as the disease is very insidious in its onset. The importance of an early diagnosis can be easily recognised, in view of the immense amount of harm that a brood mare or stallion may occasion. In neither are the symptoms at all noticeable in the beginning. A mare may infect a stallion by one act of coition and he may infect a whole district. It has been shown that the parasite may live in the vaginal mucous membrane and discharges of the female and the urethral and seminal fluids of the male for an indefinite period. The only really diagnostic test is the demonstration of the trypanosoma equiperdum.

In districts in which there is any history of the disease, all cases which show the least sign of irritation of the penis or vulva amongst stallions or brood mares should be immediately examined by taking a scraping from the urethra or vulva and submitting it to a microscopical examination. In even freshly contracted cases the parasite will as a rule be found, which will establish a positive diagnosis. Though the result may at first be negative it cannot be accepted as such. Further examinations must be made until such period as the plaques should appear. If these appear, then a positive diagnosis can be made; if not, the case is not dourine.



The temp. continued like this until the animal was destroyed on the 9th December

CHART 2.

Showing the variations in the temperature of the stallion depicted in Fig. 4.

In the event of plaques being present, a drop is taken from a freshly formed one, where the organism most surely will be found, probably in small numbers, one or perhaps two to a field of the $\frac{1}{6}$ inch objective. If the plaques are old (twenty-four hours) there may be no parasites. They tend to diminish, and disappear entirely in a short time, and even in the tissue immediately surrounding the eruption no parasites may be found. In fact the further removed from the centre the puncture is made, the smaller in number are the parasites likely to be. That they exist in the blood there is no doubt, for dogs have been infected by inoculation of blood when no parasites could be seen in the general circulation, though present in the plaque.

Differential Diagnosis.—The disease is characterised throughout by its intermittence. As each fresh crop of parasites appears in their cycle of existence, there is an exacerbation of symptoms, but, in contradistinction to the malarial hæmatozoon of man and the piroplasmo of cattle, there is no marked rise of temperature and a correspondingly low fall. I attach two typical temperature charts in which it will be seen that with the exception of the rise of temperature in the initial stage there is no marked rise and fall. I am indebted to Major Pease of the Punjab Veterinary College for allowing me to bring these charts from the College Hospital.

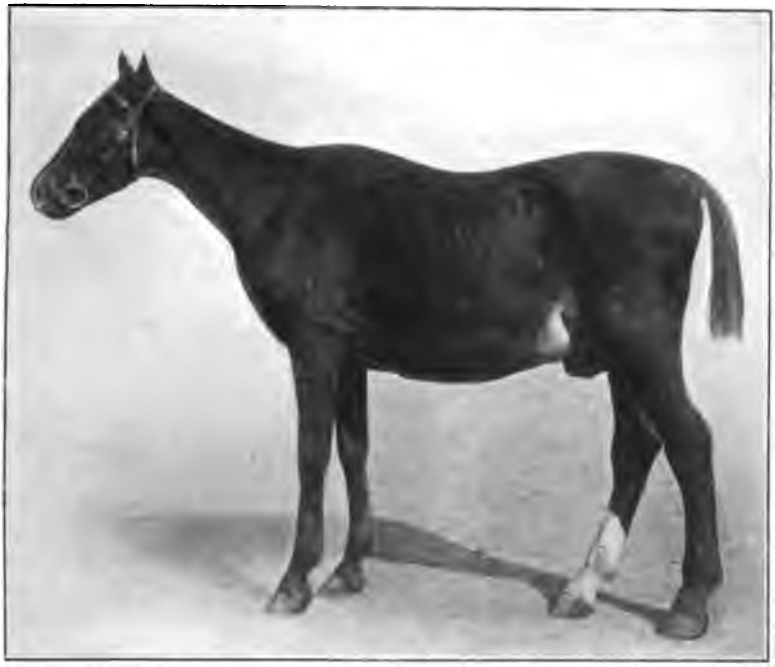


FIG. 4.

Thoroughbred English horse, in the third stage. Shows enormous œdematous infiltration under the abdomen, swelling of the hind legs, and emaciation. Trypanosomes were detected at the time the photograph was taken. Naturally infected.

The disease in some aspects may simulate glanders, but this may at once be distinguished by testing with mallein.

The trypanosomiasis due to surra is very similar, but the history of the cases would be quite different. In surra there are seldom isolated cases, and, moreover, the animal will come from a surra infected area where there are sure to be some cases among geldings. Dourine never attacks geldings naturally, or mares that have not been to the stud. The temperature chart in surra shows a very high rise and a correspondingly low fall at regular intervals. The parasite (trypanosoma Evansi) can always be found swarming in the peripheral blood when the temperature is high, and often in smaller numbers when the temperature is a little over 100° F.

The morphology of the surra organisms is different—it is larger, being from 28 to 36 microns. Its centrosome extremity is longer and sharper, and the centrosome and nucleus have not such an elongated appearance. There will be œdematous swellings, but the penis will not show ulceration or the scars of recently healed ulceration. The continuous petechial spots on the eye mucous membrane are always associated with surra, but not so common in dourine. There are never any plaques in surra.

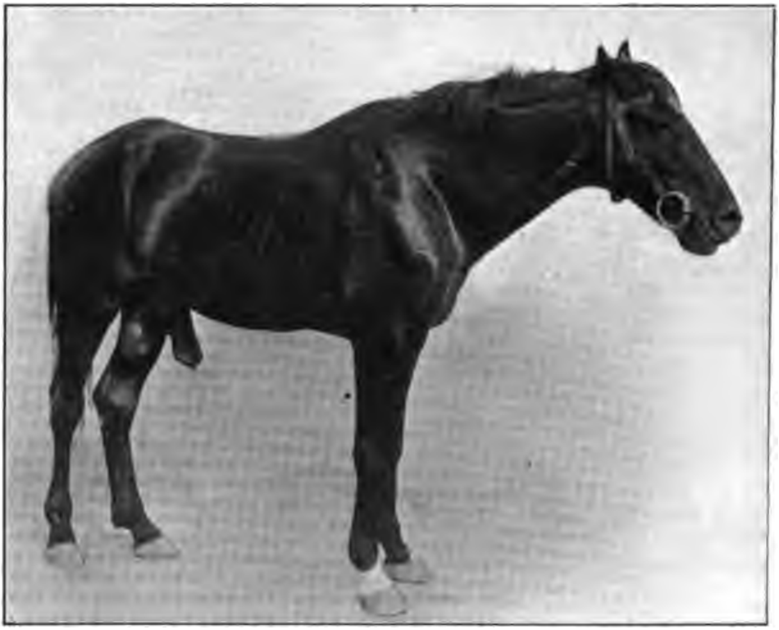


Fig. 5.

Thoroughbred English horse, final stage. Penis œdematous, tense, and partially paralysed. Depressed appearance. There was loss of co-ordination when moving, and plaques had been present in large numbers. Naturally infected.

From the experiments of Pease, a ready diagnosis can be made by the inoculation of a pariah dog (in India), which has shown quite a remarkable immunity to dourine but is very easily susceptible to surra.

In dourine, as I have already said, the temperature is never very high (100·5° to 101·5° F.) and the parasite is with difficulty demonstrated in the peripheral circulation, where it is very easily found during the paroxysm in surra. In dourine it should be sought for in the discharges from the urethra and vulva, in recent œdemata, and in the plaques. Dogs may be inoculated with blood, preferably from an œdematous area; in surra blood from anywhere suffices.

Purpura hæmorrhagica might in the early stages have some likeness to dourine, but the history and character of the swellings should easily obviate an error in diagnosis. The disease, mal de caderas, due to the trypanosoma equinum, and described in a translation from the Spanish by Capt. Barry in the *Veterinary Journal*, is similar in character to dourine, but the parasite is very different. The trypanosoma equinum has no centrosome, which is sufficiently easily demonstrated and renders diagnosis easy. In length it is about the same, but it is broader and more squat in appearance.

In the ass there is more difficulty in diagnosis, the disease being considerably more chronic at its onset and also in its course, so that there is a greater danger in its not being recognised in the earlier stages, which recognition is of so much importance with a view to prophylaxis. Pease says that it often happens in India that the first notification one has of the jack being affected is the fact that some of the mares which he has served in the district in which he is used for mule breeding begin to show signs of the disease. There is, however, observable on close examination, even in the early stage, the œdematous condition of the extremity of the penis, which is invariably present, and causes the folds of the mucous membrane at the urethral orifice to be obliterated, so that the urethra appears to be turned inside out. Other symptoms are similar to those in the horse, but there is not so much tendency to ulceration of the external genitals. The same authority says that paralysis sets in

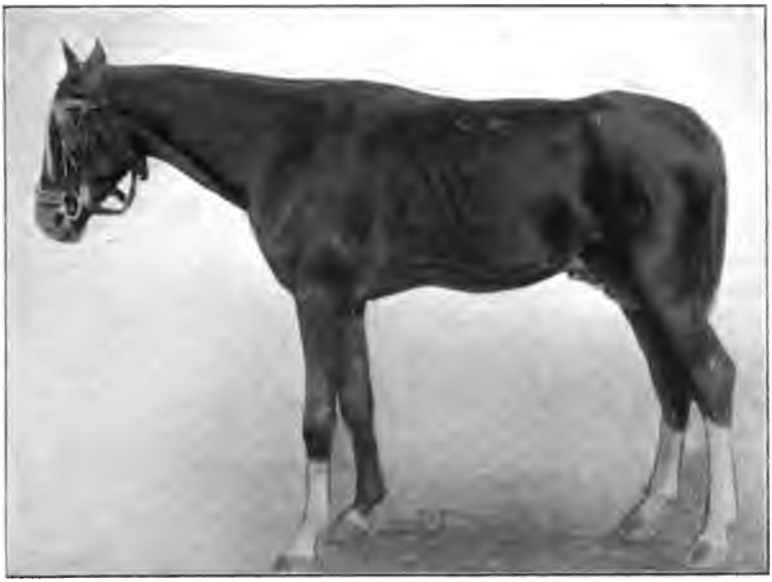


FIG. 6.

A bay Arab horse, in final stage. Extreme emaciation, but œdema not very noticeable except about the knees. Shows indifferent and sleepy aspect. Numerous plaques had appeared, the marks of some of which may be seen on the ribs. Naturally infected.

comparatively early. As in the horse, a positive diagnosis cannot be given unless the organism is demonstrated.

Morphology of the Parasite.—The trypanosome may be best observed in a stained preparation of a blood smear, and it will be seen as a rule in various stages of development, if there be a fair number present. There will be the ordinary forms already described, and in addition dividing and degenerating forms. Division takes place in the longitudinal axis only, and the macro-nucleus divides somewhat later than the micro-nucleus or centrosome. When a parasite is about to divide it becomes thicker and shorter, and then the division of the micro-nucleus follows, which becomes two. The second centrosome has extending from it an undulating membrane, terminating in a flagellum. This may extend at right angles to the original organism or in an entirely opposite direction, but usually it is parallel to the pre-existing one. In the meantime the macro-nucleus approaches the centrosome and becomes larger, and then also divides. The division proceeds until there are two distinct organisms united by their posterior (centrosome) ends only, and these finally separate. It is also possible that an organism may divide into more than two at the same time, and it is likely that this formation has given rise to the idea of the rosette form of multiplication. The simple division into two forms has also been mistaken for conjugation; as far as is at present known, no conjugation takes place in the horse's blood in any of the forms of trypanosomiasis. There are more than possible other intermediary forms in which exist a sexual cycle similar to that of the malarial parasite as it passes through the mosquito, but up to the present this has not been demonstrated.

Occasionally in specimens which are degenerating in blood that has been drawn for some time, or in old cachectic cases in which the parasites are numerous, several may be seen crowding together with their posterior ends almost in apposition. This is an attempt at agglutination, and has been mistaken for conjugation and the rosette forms of division.

Holmes, in an article on the evolution of the surra trypanosome published in the *Journal of Comparative Pathology* for September 1904, argues very well that there is conjugation of the parasite in the horse's blood. This may be as in the malarial organism, which never does so except on release from the human host. However, the fact that two organisms "meet and break away again" during their rapid movements is not sufficient evidence of actual conjugation.

Staining Methods.—The best methods of staining are Romanowsky's and Wright's modification of Leishman's method; the latter is very useful and handy, as no mixing of solutions is necessary and no fixing required.

The following is *Romanowsky's Stain*:—

Stock Solution No. 1.

Höchst's Medicinal Methylene Blue	1 part
Sodium Carbonate pure	0·5 parts
Distilled water	100 parts

Place this solution in an incubator at 37° C. for two or three days, when a purple colour will be noticed at the edges of the liquid; this depends upon the formation of a new red colour—methylene red—which combined with eosin forms the active principle of the stain and has a particular affinity for chromatin. Unless this polychromatophyllic change takes place the solution is useless.

Stock Solution No. 2.

Eosin	1 part
Water	1000 parts

For staining, the stock solutions are separately diluted with water, 5 parts of stock solution to 100 of water.

In order to obtain a smear, prick the centre of the plaque and take a drop of blood on a slide, which should be chemically clean, having been taken from an alcohol bottle and dried with a clean piece of art muslin. Then, either in the ordinary way with a piece of cigarette paper, make a thin even smear over the slide, or, as an easier and equally efficient method, take a perfectly clean flat large-sized needle and place it edgeways on the drop, when the capillary attraction of the needle will cause the blood to stream right across the slide; then

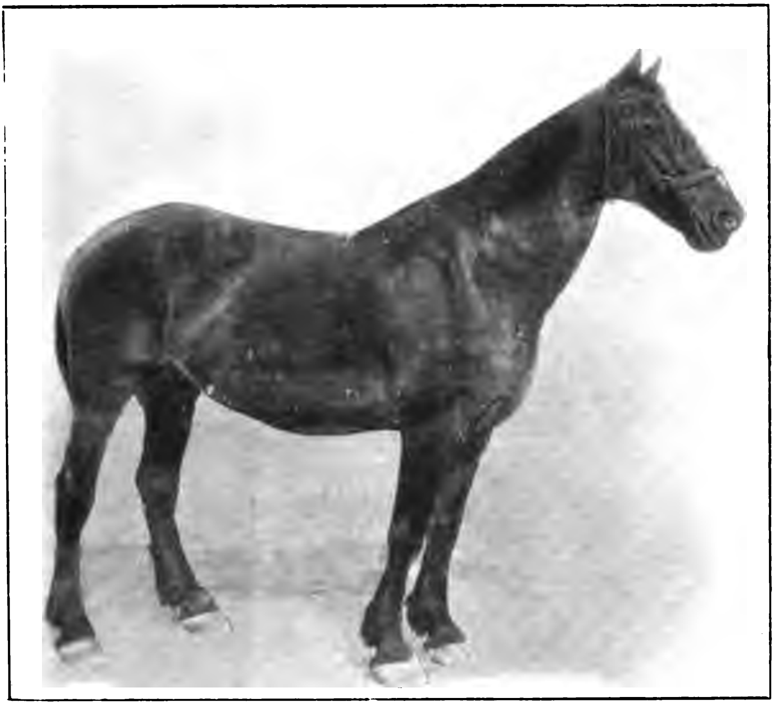


FIG. 7.

A bay Australian mare. Acquired disease by inoculation with infected material on the mucous membrane of the vagina. Now at the beginning of the cuticular stage, but has not yet shown any constitutional disturbance. Plaques had appeared, and the marks of some of them can be seen on the neck and shoulders, as the hair was shaved from each when blood was withdrawn. At this time numbers of trypanosomes were present in the vaginal mucus. There is very slight œdema of the hind legs.

evenly and gently draw the needle down the length of the slide, and a very even smear may be obtained. Quickly dry the smear in the air by waving rapidly about, which prevents the red corpuscles from crenating; the slide can then be kept indefinitely or used at once. The advantages of using a slide instead of a cover-glass are that you get a much larger field on which to work, it is much more easily manipulated, and it can be kept without any mounting. Place the film in absolute alcohol or alcohol and ether, for fifteen minutes to half-an-hour, to fix. This coagulates the albumen and makes a permanent film in which the corpuscles and organisms are retained. Remove from the alcohol, wash in water, and then apply the stain. This is made by mixing equal parts of the above two solutions, freshly prepared, in a small glass measure or porcelain dish. It is important that the admixture should be as fresh as possible. Apply the stain to the whole of the film and let it remain for seven to ten minutes; wash in water and dry in the air, no heat being applied. The red corpuscles may have a bluish tinge, which may be removed by further washing. If the blood platelets appear bluish the film requires further staining; they should appear as ruby-red granular bodies. By continuous application of water the stain may be washed out, but the film may always be stained over again.

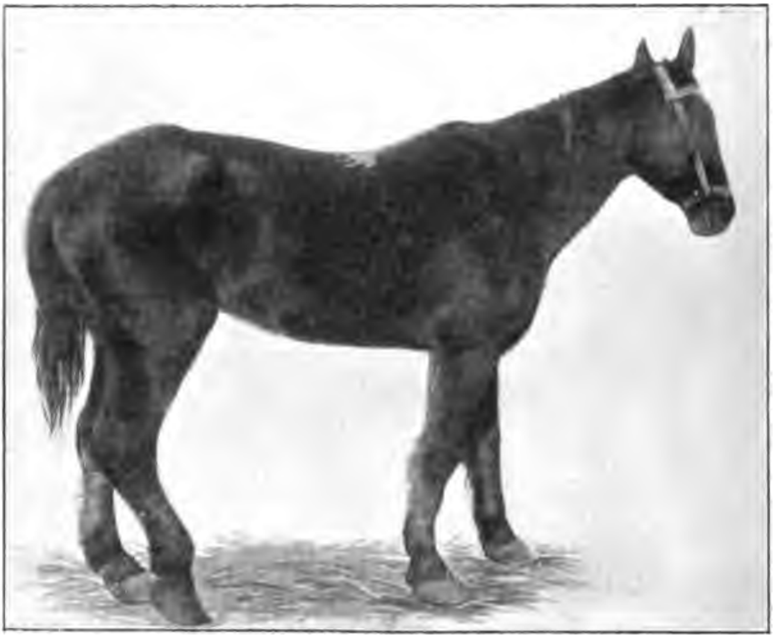


FIG. 8.

A bay Australian mare. Acquired the disease by subcutaneous inoculation. Is now in the final stage and shows the same listless indifference manifested in the stallions. Rapid emaciation and œdema of the hind limbs, especially the off one. This mare showed symptoms of arthritis, as is indicated by the posture. Plaques had been very numerous.

By this stain the protoplasm of the trypanosome is stained blue, the nuclear chromatin a carmine violet, and the flagellum and centrosome a brilliant red. The red corpuscles will be a pinkish colour, and the various forms of leucocytes will be well differentiated. In examining the smear time may be saved by looking along the edge of the film, as it is here that the parasites will be most numerous if they are present, as they, being like the leucocytes of less density than the rest of the blood, tend to run to the periphery when the smear is made.

A film made in this way requires no cover-glass, but if the cedar oil is left on it tends to withdraw the colour; however, if it be carefully blotted and wiped off with a soft rag after use the film may be kept indefinitely.

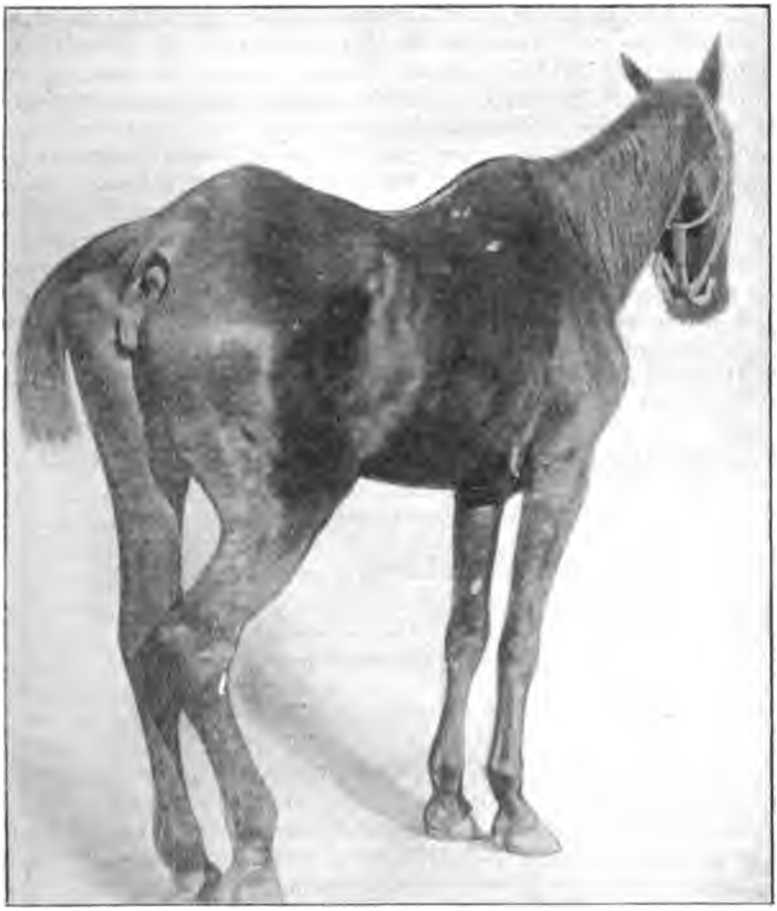


FIG. 9.

Bay Australian mare. Acquired the disease by rubbing of infected material into the nasal mucous membrane. Now at the beginning of the final stage. Shows marked emaciation and sinking in of the vulva and anus, which is characteristic of this period. Some plaques had occurred. There is slight œdema of the hind legs, and the off one shows the effect of the serous discharge on the hair.

Leishman's Stain is a modification of Romanowsky's, and consists of a product of the interaction of eosin and polychromatic methylene blue dissolved in methyl alcohol.

Dry the film and pour on sufficient solution to cover it (*vide* preparation of Wright's stain). No fixing is necessary, as the methyl alcohol acts as a fixing agent. Allow it to stand for half a minute. Add about twice as much distilled water and rock the slide. Allow it to stand for five minutes longer. Then wash in water for a few minutes. This intensifies the stain, removes the deposit, and changes the colouring of the red cells from greenish to faint pink.

Wright's Stain is a modification of Leishman's, and, like that, it is very useful for quick work. It is applied directly to the dried film and allowed to act from one to three minutes. A few drops of distilled water are added until a metallic scum is formed; after a few minutes the slide is washed with distilled water, and then dried in the air. Prepare as follows:—

Take 100 cc. of distilled water and dissolve in it half-a-gramme of sodium bicarbonate. Then add 1 gramme pure methylene blue and steam for one hour. Cool. Make a solution of eosin in distilled water, half-a-gramme to 500 cc. Add this solution to the methylene blue solution with constant stirring. If the eosin is correct in amount a fine metallic scum will form on the surface and a precipitate be formed in the body of the liquid. This precipitate is a combination of eosin and methylene blue. Recover this by filtration and then dry. Dissolve three-tenths of a gramme in 100 cc. of methyl alcohol. Filter and add 20 per cent. more alcohol, and it is now ready for staining.

Ziemann's Modification of Romanowsky's Stain is as follows:—

Stock Solution No. 1.

Methylene Blue	1 gramme
Borax	2·5 grammes
Distilled water	10 grammes

Stock Solution No. 2.

Eosin	1 gramme
Water	100 grammes

The solutions are mixed immediately before use in the proportions of one part of the No. 1 solution to four parts No. 2 solution.

Technique the same as in Romanowsky's method. The differentiation is similar to Romanowsky, except that the chromatin of the centrosome is a deeper red than the flagellum.

The trypanosome may be stained by carbol-thionin or methylene blue, when its characteristics may be well seen but not differentiated as to colour. The entire organism will be stained blue, but the chromatin of the centrosome shows greater affinity for the stain and is more deeply coloured.

Laveran and *Mesnil's Stain* is more complicated. A few silver nitrate crystals are placed in a 150 cc. phial with 50 cc. of distilled water, and when the crystals are dissolved the flask is filled up with a saturated solution of carbonate of soda and shaken repeatedly. A black precipitate of oxide of silver is formed, and this is washed several times with distilled water so as to remove the nitrate of silver and excess of soda. Then pour upon the oxide of silver a saturated aqueous solution of Höchst's medicinal methylene blue. Leave in contact for two weeks at ordinary room temperature, shaking repeatedly. This forms Borrel's Blue. To make the stain take:—

Borrel's Blue	1 cc.
1 per mille Eosin Solution . . .	4 cc.
Distilled water	6 cc.

Prepare and fix the smear in the usual way, as above described. Stain with above for twenty to thirty minutes. Wash freely with water and then with distilled water. The film may now be examined, or it may first be treated with a 5 per cent. solution of tannic acid for ten to fifteen minutes, washed thoroughly, dried, and then examined. If there is any haziness wash in clove oil, then in xylol, dry, and examine. Smears by this method are best examined unmounted, and treated as already described.

Mortality and Loss.—Dourine is a most fatal and incurable disease except in the initial stage. From statistics collected during various outbreaks in Germany and Russia, the mortality would appear to be at least 50 per cent. of those attacked. In India I have never seen a stallion recover that has passed the initial stage.

Pease says that of mares exposed to infection in India at least one-third or possibly more become infected, and of these 70 to 80 per cent. die. I have seen cases of recovery in stallions which, at the first appearance of œdema and genital lesions, I had promptly castrated, but in some cases in which the operation was delayed no recovery took place. I am not aware what the beneficial effect of castration is, but it does in some cases successfully check the disease.

If the disease has progressed to the second stage such treatment will be of no avail. In addition to castration, arsenic in full doses, combined with nux vomica, potassium iodide, and general tonics may be tried. The general health is to be kept as perfect as possible, and occasional salines ought to be exhibited. A liberal diet and strictly hygienic conditions are important. Local ulceration will yield to ordinary antiseptic dressings.

The chief effort, however, in connection with the disease should be directed to prevention.

Prophylaxis.—In Austria, where the disease has been existent for a long time, the following rules are adhered to, and I think they sum up the necessary preventive measures. It is understood that they apply to a country in which the disease is prevalent:—

(1) Even when there is nothing to lead to the supposition that the disease exists, every mare about to be put to the horse shall be carefully inspected, and refusal made to old and weakly mares, or to those which have a discharge from the vulva, or have that organ enlarged or swollen, or which do not present the ordinary manifestations of œstrum. It is also suggested that an œdematous swelling, no matter where situated, should negative covering.

(2) The stallion's penis to be carefully and frequently examined, and on no account is the animal to be used if there is the slightest lesion upon it. He is to be kept secluded until all doubt as to the nature of the lesion has passed away.

(3) Give every information possible to breeders as to character, etc., of the disease.

(4) Immediate information is to be given in all cases of stallions in the least suspected, and the necessary steps taken.

(5) To prevent extension, the sale of all mares in the affected areas to be stopped during the prevalence of the disease.

(6) If the malady has spread in a district, all breeding stallions to be stopped employment, whether Government or private property. Those already diseased to be sequestrated under police supervision, whether private or Government proverty.

(7) Affected animals to be separated from healthy, to have their own attendants, and no interchange of clothing, utensils, etc., to take place. Those deemed curable to be treated, those incurable to be destroyed.

(8) All horses attacked to be castrated, as well as those which, notwithstanding their apparent good health, have transmitted it to mares they have served, and also those which have been put to infected mares.

(9) Mares that have been in the least affected and apparently cured not to be covered the following year, or until certified by a veterinary surgeon as cured. It is even better to exclude all such mares entirely, and brand them as having had the disease.

The disease being conveyed solely by coitus, it should not be a difficult matter to stamp it out, especially in India, where all, or practically all, the entire horses are Government property. This can be effected by the employment of sufficient veterinary surgeons to superintend the systematic examination of all stud horses and mares. The only difficulty lies in the fact that many stud horses are situated singly in isolated districts, with no one having any expert knowledge in charge except a native groom. They can only be visited very occasionally by the veterinary officer, and mares that are brought to the horse to be covered would not be detected by the groom as in any way diseased, unless they were very bad. This difficulty, however, could be overcome by concentrating horses in bigger depôts, with an expert subordinate in charge, and by segregating infected districts, and withdrawing isolated stallions from such districts.

Special care is required in the periodic examination of jack donkeys used for mule breeding.

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THE PROPHYLAXIS OF GLANDERS.

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GLANDERS is essentially a disease of the horse, and in considering the means which are appropriate to its prevention or extermination only the facts relating to equine glanders need to be taken into account. No doubt there are certain precautionary measures which ought to be enforced with the object of preventing the infection of human beings from glandered horses, but the true prophylaxis of the disease in man lies in dealing with the infection at its fountain-head, viz., in the horse.

I propose in this article to pass under review, (1) the extent to which the disease at present prevails in this country, (2) the methods by which it is disseminated, (3) the prophylactic measures which have been opposed to it, and (4) the reasons why those measures have hitherto proved unsuccessful, and the alterations which ought to be made in them.

The statistics published by the Board of Agriculture show that thirty years ago, viz., in 1874, 636 cases of glanders in the horse were reported in Great Britain, and since that year the annual total has never been so low. In 1879 it was 1367, and in the following year it leapt up to 2110. After that there was a gradual decline, until in 1885 the total cases reported numbered 946. The next seven years was a period of increase, until the annual total had risen to 3001 in 1892. Again there was a period of diminished prevalence, lasting till 1898, when the total was 1472. From that year up to the present time has been another period of recrudescence, and last year, with a total of 2658 cases, was the worst since 1892.

The facts relating to the geographical distribution of glanders in Great Britain are very striking. Although as a rule cases are reported annually from between thirty and forty counties, the county of London has always been the great centre of the disease, and during the last five or six years it has contributed about 75 per cent. of all the cases reported. Of the remaining 25 per cent. as a rule not less than half is contributed by the counties immediately around London, viz., Middlesex, Kent, Essex, and Surrey. Thus, nearly 90 per cent. of all the reported cases of glanders are furnished by the Metropolitan area. Liverpool, Manchester, and Birmingham furnish a notable proportion of the cases occurring in England outside this area; Wales is nearly exempt from the disease; and of the outbreaks reported from Scotland more than 50 per cent. occur in Glasgow and Lanarkshire.

What these figures indicate quite clearly is that glanders is now almost entirely a disease of London and the other large cities. In these it has probably existed continuously for centuries, and at the present time they constitute what may be called the breeding-places of the disease. The outbreaks occurring outside the large towns are, from the prophylactic point of view, unimportant, as they are practically all due to the importation of diseased horses from London or some of the other cities previously mentioned.

In dealing with these statistics it is important to remember that for the most part they relate to what are now generally called clinical cases of glanders, that is to say, horses which were slaughtered in consequence of the development of outward signs of the disease, and do not afford much information regarding the number of horses actually affected with glanders. That is a fact of the highest importance, and it is one which has only been apprehended within the last dozen years. Prior to that time the prevalent view undoubtedly was that glanders ranked among the most virulent diseases of the horse, in the sense that it soon provoked manifest signs of illness, and in most cases ended fatally when allowed to run its natural course. It is unquestionable that that was a wrong view of glanders. It is only in a minority of cases that the act of infection is early revealed by the onset of symptoms of ill-health or the development of external lesions, and in many cases infected and actually diseased horses retain for months or years every outward sign of good health.

Moreover, it is now impossible to doubt that when affected horses are kept under good hygienic conditions, and especially when they are well fed and not over-worked, a considerable proportion recover from the disease without having at any time shown any symptoms of illness or developed any external lesions.

For a knowledge of these two important truths we are indebted to the use of mallein. Resort to that as an aid in diagnosis soon showed that a pronounced reaction was often obtainable in a considerable proportion of the apparently healthy horses in infected studs, and by *post-mortem* examination of apparently healthy animals which had thus reacted there was soon accumulated sufficient evidence to prove that what may be called latent glanders is very common in large infected studs. In like manner the negative results of subsequent tests with mallein at long intervals, coupled with the negative results of the *post-mortem* examination of horses which had thus ceased to react, gradually proved the second point, viz., that a good many horses make a complete recovery from glanders. The truth appears to be that occult glanders is the rule and not the exception, for even in those cases in which the disease becomes so acute that the horse's life is threatened, it is probable that a period of months often elapses between infection and the onset of distinct symptoms. This is the fact which gives the clue to the spread of the disease. No doubt some fresh outbreaks are due to the introduction of cases of glanders which are more or less clinical, but in the great majority of fresh outbreaks the infection is introduced by a horse in which the disease is latent. A question that has been much discussed in this connection is whether a horse affected with latent glanders is capable of communicating the disease, or only becomes dangerous when outward signs of the disease are developed.

I think the balance of argument is all in favour of the view that no horse affected with glanders can be considered incapable of spreading the disease, and that many horses discharge the specific bacilli and thus infect their neighbours while the disease is still in the occult stage. To maintain the contrary appears to me to be equivalent to asserting that the disease is never spread except by horses affected with farcy, or with glanders associated with an obvious nasal discharge resulting from ulceration, for the only other outward condition at all indicative of glanders is chronic enlargement and induration of the submaxillary lymphatic glands—a lesion which, except in those very rare cases where the glands suppurate, adds nothing whatever to the horse's powers of infection. It may be freely admitted that by far the most dangerous cases are those in which a nasal discharge or farcy lesions are present, but it is hard to believe that glanders bacilli never find their way outwards from the pulmonary lesions of so-called occult cases.

In truth, however, this question has far less practical importance than might at first sight appear. The horse that is affected with latent glanders to-day may be non-infective, but he will certainly be infective when he develops a nasal discharge or farcy lesion, and in practice it is impossible to ensure that the horse will be isolated the moment he thus becomes highly dangerous.

Let us now see what are the measures which the law enforces with the object of preventing the spread of glanders. These measures are prescribed in the Glanders and Farcy Order, 1894, of the Board of Agriculture, and the execution of them is in the hands of the Local Authorities under the Diseases of Animals Act.

In the first place, the Order lays on the owners of glandered animals the duty of notifying the fact to a constable of the local police force, who in turn must convey the information so received to an Inspector of the Local Authority. The Inspector must then proceed with all practicable speed to the place where the disease exists, or is suspected to exist, and there and elsewhere put in force and discharge the powers and duties conferred on him by the Order.

In the event of the case being considered one of glanders or farcy by the Inspector, the Local Authority may, if they think fit, cause the animal to be slaughtered, provided the owner does not object. In the latter case the animal cannot be slaughtered without the special authority of the Board of Agriculture. For any diseased horse slaughtered the Local Authority must pay compensation, the minimum being fixed at two pounds, and the maximum one-fourth of the value of the horse immediately before it became diseased.

The Order also impowers the Local Authority, but only with the consent of the owner, to slaughter any suspected animal, and, in the event of its proving to have been diseased, to pay compensation within the limits just specified.

Article 6 of the Order empowers any Local Authority to make such regulations as they think fit for prohibiting the movement of diseased or suspected animals, or of animals which have been in contact with diseased animals or otherwise exposed to infection, but the movement of the latter class of animals can only be prohibited when the building or other place to which the restriction applies contains a horse which is held to be actually diseased.

Article 9 makes it illegal for any person to expose diseased or suspected horses in public places, to graze such animals on pasture on the sides of a highway, or to carry, lead, or drive them on a highway or thoroughfare.

The Order contains other articles dealing with disinfection, seizure of diseased or suspected animals exposed in contravention of Article 9, etc., but the only provisions of the Order which are really important for an understanding of the effects which it is calculated to produce in the way of stamping out the disease are those of which the sense has just been given.

Now, as the statistics already quoted prove, this Order has absolutely failed to exterminate glanders, and is even ineffectual to prevent the disease from extending. The reason for this failure is not far to seek. It lies in the permissive character of the Article which deals with the movement of diseased and suspected horses, and in the interpretation which in practice has been placed on the words "diseased" and "suspected." In exercising the powers conferred on them by the Order, Local Authorities have hitherto acted on the principle that no horse is diseased unless he shows external symptoms and lesions which in the opinion of the Inspector are characteristic of glanders. Practically this means that before the horse can be condemned and dealt with as diseased he must develop either farcy lesions or nasal ulceration. Although a horse may be known to have been exposed to infection and have reacted to mallein, he is not in practice treated as a diseased horse. Nevertheless such a horse is really more certainly glandered than many which are condemned because they exhibit external lesions supposed to be indicative of glanders.

Turning next to the interpretation of the word suspected, it has to be noted that Article 6 of the Order draws a distinction between "suspected" horses and horses that have been "exposed to infection." If the latter are not to be reckoned suspected, one wonders what exactly is meant by the term. In practice it appears that a horse is called "suspected" when it exhibits external symptoms, but these leave the Inspector in doubt as to whether the case is one of glanders or not.

It might be thought that Article 6 of the Order would enable Local Authorities to deal effectually with outbreaks of glanders, since it empowers them to prohibit the movement of horses which have been exposed to infection, but it will be remembered that the movement of a horse cannot be prohibited by a Local Authority unless it is the stable companion of a diseased, that is to say, clinically glandered, horse.

Finally, in considering how the Order is worked, it ought to be noted that it does not empower a Local Authority to test suspected horses with mallein.

Here, in London, what is called an outbreak of glanders is usually dealt with as follows. When a case of glanders is notified, or in any other way comes to the knowledge of a Veterinary Inspector to the Local Authority, it is visited by the Inspector, and, with the consent of the owner, it is slaughtered, the minimum compensation allowed under the Order, viz., £2, being paid. If none of the other horses in the stable are clinically glandered, nothing more is insisted upon except disinfection of the stable and destruction of the litter. When that has been done to the satisfaction of the Inspector the outbreak is considered at an end. As a matter of fact it very rarely is at an end, for this procedure almost invariably leaves one or more cases of occult or latent glanders in the stables. If these remain in the possession of the same owner, some of them will later on become clinically glandered, and, if the fact is notified, they will figure in subsequent outbreaks. But the owner is perfectly free to sell horses against which there is a suspicion owing to there having been exposed to infection, and many such horses are sold every week in London. Indeed, there is reason to fear that worse than this sometimes happens. When glanders breaks out in a stud, the owner may sometimes resolve to sell all the apparently healthy animals as the only certain way of getting rid of the disease, and that, although a legal, is not a strictly moral, proceeding, but it is positive virtue compared to the practice of testing the whole stud in which an outbreak has occurred and selling the animals that react. It would probably be a libel on horse-owners to say that this is frequently done, but, as previously stated, the sale of horses to which a suspicion of glanders justly attaches is a very common proceeding.

The great defects in the existing regulations with regard to glanders are (1) that they ignore the great service which mallein could render in diagnosis, and (2) that they do not deal in a serious manner with the horses known to have been exposed to infection. For years past this has been well known to all the Local Authorities in whose districts cases of glanders commonly occur, and there is now practical unanimity as to the amendments which ought to be made in the existing Glanders and Farcy Order.

In the first place, the word suspected must be given its natural interpretation, and made to cover all the apparently healthy horses in every stable in which a case of glanders has recently occurred. If this slight alteration were made in Article 6 of the Order it would enable the Local Authority in every outbreak to confine the whole of the reasonably-suspected horses to the stable, until they are able to ascertain which of them are really infected. While the horses are alive, there is only one way by which this information can be obtained, *viz.*, by subjecting the whole of the suspected animals to the mallein test. There does not appear to be any valid reason why the Board of Agriculture should not recognise the remarkable reliability of the mallein test, and empower Local Authorities to use it on all suspected horses. The Departmental Committee which was appointed to consider the subject of glanders in 1899 thought otherwise. They reported that mallein was "a very sensitive diagnostic reagent," but, nevertheless, they came to the conclusion that they could not recommend that the use of it should be made directly compulsory. This, however, did not prevent them from recommending that the Order should be so altered as to make it indirectly compulsory. The plan which they favoured was to empower Local Authorities to treat "in contact" horses as suspected, that is to say, to confine them to their stables, until the owner consented to their being tested with mallein. Presumably it was thought that to indirectly enforce the use of mallein would excite less opposition than to directly prescribe it for all suspected animals. If so, the Committee probably did an injustice to the intelligence of horse-owners. The next alteration required in the Order is one to empower Local Authorities to regard a horse which reacts to mallein as a glandered horse. It does not follow, however, that the apparently healthy horse that has reacted to mallein ought to be regarded as glandered in the same sense as a horse affected with farcy or nasal ulceration. He is in a different category, (1) because he is generally far less dangerous, and (2) because he is much more valuable. As a rule, what is called a clinically glandered horse is valueless, because he is incurable and, unless strictly isolated, a source of great danger.

Even when isolated he is a source of danger to the man who has to attend to him. On the other hand, an apparently healthy horse which is only known to be glandered because he has reacted to mallein, has still a certain value. His working powers may at the time be unimpaired, and he may even make a complete recovery. It is true he would be of very little value if, in spite of his physical fitness for work, it were necessary in the public interest to maintain him in a state of isolation. This latter is a very important point, because what ought to be done with apparently healthy reacting horses depends on it. The Departmental Committee of 1899 considered this question, and in their Report they expressed the opinion that, while such horses were probably not so infective as clinically glandered horses, it was desirable to consider them "as possible sources of danger." In view of its importance, the matter was referred to a second Departmental Committee which was appointed by the Board of Agriculture in 1901, and this Committee, after carrying out a series of experiments with horses that had recently reacted to mallein expressed the opinion that the danger of glanders being spread by apparently healthy reacting horses while at work in the streets is so slight that in practice it might be neglected. As the result of other experiments with horses that had at one time reacted to mallein, and had after a considerable interval ceased to react, this Committee also reported that in their opinion such horses were incapable of spreading the infection of glanders.

Assuming that these opinions are correct, the public interest does not demand that reacting but apparently healthy horses should either be slaughtered or prevented from working on the streets or highways. On the other hand, the interests of the owner himself demand that such horses should not be stabled with healthy horses, and in the public interest it is necessary to insist that they should not be allowed to change hands. No doubt the public interest would be best served by their prompt slaughter, but equity demands that where that is enforced the owner should receive reasonable compensation.

To sum up then, it may be said that the Glanders and Farcy Order now in force has failed because it does not empower Local Authorities to deal adequately with the horses which in nearly every outbreak are legitimately suspected through having been in the same stable with a glandered animal. Glanders will never be stamped out until such exposure to infection is viewed as a sufficient reason for regulating the movement of the horses concerned. In every outbreak unless the whole of the animals are clinically glandered, or the owner consents to their being so regarded and slaughtered, the use of mallein should be imposed by law. In most outbreaks that will clear all but a small minority of the horses from suspicion. In the public interest the horses which have reacted must be so dealt with as to prevent them from spreading the disease. That might be secured in some cases by having such animals branded, and allowing them to be used for work on the condition (1) that they should not be stabled with healthy horses, and (2) that they should not be allowed to change hands, but be finally slaughtered under police supervision.

Perhaps this last condition might be so far modified as to permit the sale, or the removal to a healthy stable, of horses which have ceased to react. Personally, I am inclined to think that it would be better to forbid both of those procedures, although I do not doubt that many reacting horses make a genuine recovery, and that failure to react after an interval of several months is fairly reliable evidence that this has actually happened.

The merit of this system lies in the fact that it would enable outbreaks in large and badly-infected studs to be dealt with without seriously dislocating business and without imposing on Local Authorities the task of finding large sums to be paid in compensation. In many small studs it would, on the other hand, be impracticable, and it would probably excite a good deal of opposition on the part of people who cannot reconcile themselves to the notion that horses known to be glandered should be allowed to work on the streets, although it is, I believe, quite certain that thousands of horses so affected are working in London every day.

Undoubtedly a better but more expensive method would be to compel the use of mallein on all suspected horses, and promptly slaughter all those that react. This would be an expensive method, at least at the outset in London, because it could not be enforced without allowing fairly liberal compensation, and because there is great reason to fear the number of animals which would have to be slaughtered would be a large one. The Departmental Committee of 1899 recommended somewhat vaguely that the compensation to be allowed for the slaughter of reacting but apparently healthy horses should be "on a higher scale" than for a clinically diseased horse, but two members of the Committee were in favour of the amount being fixed at half the value of the animal before it became diseased. On an average this would probably work out at about £20 per head. Such a sum would not be too liberal, but it is much to be feared that the aggregate necessary to meet the claims for compensation, even under this scale, during the first year would reach a very formidable figure.

During the last five years over 2000 horses have been slaughtered annually on account of glanders, and nearly the whole of these were cases of clinical glanders. If the studs from which these cases came had been tested with mallein it is probable that not less than five times as many would have reacted, or say 10,000 horses, which, at £20 per head, would require £200,000 for compensation, and nearly the whole of this sum would fall on the Local Authority of the County of London. The case, however, appears to be one in which part if not the whole of the cost of compensation ought to be borne by the Imperial Exchequer, and although the extermination of glanders can only be purchased at a formidable price it ought to be remembered that the disease is at present causing an annual dead loss of not less than £50,000. Lastly, it ought not to be forgotten that the stamping out of equine glanders is the only way by which glanders in man can be entirely prevented.

The reports of the Registrar-General show that on an average glanders causes the death of about four persons annually in England and Wales (seventy-eight deaths during the twenty years 1883-1902). Considering, however, that the symptoms and lesions of glanders and farcy in man are by no means pathognomonic, and that the disease may readily be confounded with the morbid conditions which are usually classed under the heading of pyæmia, it is quite possible that the official returns considerably understate the annual mortality from glanders.

As might have been expected, the classes which contribute nearly all the cases of human glanders are ostlers, grooms, and coachmen, and the usual method of infection is by the application of farcy pus or nasal discharge to a cutaneous wound or abrasion. Whether infection can take place through the unbroken epidermis appears doubtful. In some cases the bacilli may penetrate through the nasal mucous membrane, and it is also possible that through soiling of food with dirty hands infection by way of ingestion may occur. The comparatively small proportion of persons attending to glandered horses who die from the disease has been cited as evidence against the view that inhalation is the common method of infection in the horse, but it ought to be remembered that Babes found at the *post-mortem* of a number of persons who had to do with horses, and who died from diseases other than glanders, encapsulated glanders nodules in the internal organs, and especially in the lungs,[1] a fact which would appear to suggest that occult glanders may occur in man as well as in the horse, and also that infection by inhalation may be possible in the human subject.

A remarkable fact in connection with human glanders is the almost complete immunity enjoyed by those who might have been supposed to run the greatest risk of infection, viz., the knackers and horse-slaughterers who have to deal with the carcases of glandered animals. During the last twelve years from 800 to 2000 glandered carcases have annually been dealt with in London by Messrs Harrison, Barber & Co., and I am informed that during that period no case of glanders has occurred among the men whom they employ in this work. This cannot be ascribed to any precautions which are taken to guard against infection by the men themselves, and their escape appears little less than wonderful when it is remembered that from the nature of their work they must frequently have wounds or abrasions about their hands or arms. The real explanation of their escape lies in the fact that in equine glanders the carcase in general, and the blood in particular, are usually not infective, the bacilli being almost entirely confined to the lesions and the discharge from them. The living glandered horse is far more dangerous than the dead one, and the most dangerous are those with profuse nasal discharge and the subjects of farcy. Probably the act of infection in man most frequently occurs during the administration of a ball to a horse whose mouth and pharynx contain glanders bacilli liberated from ulcers in the nose, pharynx, larynx, or trachea. One is tempted to say that when a groom or other servant contracts glanders in this way a verdict of culpable homicide ought to be brought in against his employer. In reality, however, it would be useless to advise that a ball should never be administered by hand to a horse with a nasal discharge until it has been ascertained that the case is not one of glanders, for the immense majority of horses with a discharge from the nose are not affected with glanders, and, human nature being what it is, one can easily understand the failure to recognise the exceptional case when it presents itself. It is therefore hopeless to expect that glanders will cease to figure in the returns of the registrar-general before the equine disease has been stamped out.

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[1] Article Glanders, by G. Sims Woodhead, in Allbutt's "System of Medicine," Vol. II. p. 521.

A CONTRIBUTION TO THE STUDY OF THE DISEASES OF THE EYE IN THE DOG.

By GEOFFREY H. LIVESEY, M.R.C.V.S., Hove, Sussex.

FOR the last few years I have been making a point of looking into the eyes of every dog that has come under my notice, and in many instances making a careful examination of them with the ophthalmoscope if necessary.

After seeing several thousands I have been very much struck with the frequency with which one meets the so-called "Senile Cataract," especially in medium-aged dogs, in which senile changes should not yet have commenced. Next to cataract I have most frequently seen glaucoma, and less often pigmentary retinitis. It is with these three conditions that I wish to deal more particularly.

Glaucoma is far more common than is generally supposed. By this term I mean a pathological condition of the eye in which there is increased tension of the ocular globe from increased pressure of the fluids within it. Originally the name was used (as the word signifies) for the peculiar bottle-green colour sometimes seen in the eye in this condition. But this is by no means always to be seen. It is rarely seen in man, and in only about one case in twenty-five have I seen it in the dog. When it is present, it is remarkable at once and can scarcely be overlooked. Ordinary glaucoma is, however, generally overlooked, and it is only by careful examination that its presence is detected. The disease affects one or both eyes and tends to run a chronic course, producing permanent changes in the eye and eventual blindness.

The remote causes of the condition seem rather obscure in the dog, but the direct cause is always the partial obstruction of the filtration area and consequent prevention of transudation of fluids into the Canal of Schlemm. Increased bulk of the lens or any other such cause as would tend to push the iris forward would produce more or less closure of the angle in which this filtration area is situated. Such a condition naturally tends to increase, and with it the internal tension of the eye. Sometimes cases have been brought to me because of a slight opacity of the cornea, which is often present in the early stages of glaucoma in the dog. This opacity is almost always in the deeper layers of the cornea, and must not be confused with the opacity resulting from conjunctivitis. It is nebular or slightly striated and generally situated centrally, and no doubt arises from the mechanical interference with nutrition. This condition is worth examining and becoming acquainted with, as the usual atropine lotions (so commonly supposed to be the panacea for all eye troubles) are here inadmissible. The dilatation of the pupil would only cause greater obstruction to transudation, and the condition of the patient would only be aggravated. In many cases, however, this opacity of the cornea may be totally absent, and the eye appear quite clear and bright. The dog if carefully watched may occasionally show pain by passing his forepaws over his eyes and nose, or rubbing his eye on the carpet or on his bed. There is often photophobia, and the eyes in the early stages are more sensitive to changes of temperature. The only way to test the amount of extra tension present is by palpation, but this requires a great deal of practice before it conveys much to the average touch. The eye should be gently passed back into the orbit with one finger (the lids being, of course, closed), and the hardness or elasticity of the globe tested with another finger.

When the condition is more or less acute from inflammatory changes taking place in the eye, it is easily noticed. The eyeballs may appear larger, the cornea sometimes seems more convex and may or may not be cloudy, the pupil is dilated, and the iris is pressed forward and may be almost touching the cornea. The pupil may appear brilliantly green, and there is always a marked degree of blindness. In both the acute form and the more slowly progressing the pupil is always more dilated than normal and more or less irresponsive to bright light. A healthy pupil in a young dog should contract under the influence of bright light almost to the size of a pin point, but in these cases it contracts slowly and to a far less extent. Using the ophthalmoscope, one almost invariably sees some change in the retina, the most common being pulsation of the veins, and fibrous patches over the surface and especially in the neighbourhood of the disc, with atrophy from pressure.

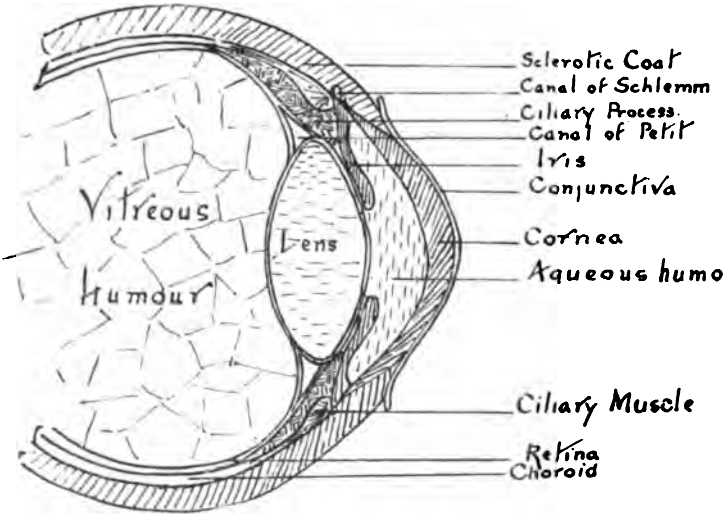

Diagram of eye in glaucoma.

The above symptoms are entirely consequent upon and invariably followed by increased ocular tension. In the first place there is interference with the circulation and engorgement of the veins, and in the second place symptoms arising from pressure on the nerves. Filaments of the fifth nerve atrophy, causing eventually diminished sensibility on the surface of the eye. The function of the third nerve is interfered with, and the ciliary body and processes are affected. The lens is relaxed, and from its normal position bulges forwards towards the anterior chamber, thereby increasing the already high tension, as has been described above. The sphincter pupillæ is also relaxed, causing wide dilatation, and hastening the course of the disease. The retinitis is consequent on the pressure, and is best seen where the nerve enters the eye. Here the disc is depressed and cup like. This condition is best seen in the acute stage.

Glaucoma defies all medicinal treatment, though eserine is of distinct value as a palliative. If taken in hand early, the disease may be at once arrested and the sight more or less preserved by the simple operation of sclerotomy, which unless carelessly performed is quite without danger or bad after effects.

The so-called senile cataract I believe to be one of the results of long standing glaucoma in a large number of cases, even though the glaucoma is only slight in degree. The lens consists of a hard nuclear portion, and a softer external portion named the cortex, which is surrounded by endothelium lining the capsule. In the substance of the lens exist minute stellate clefts. Nutrition (the lens being non-vascular) is by transudation of fluids from surrounding tissues. It is not yet clearly known how waste products are excreted, but probably they are driven inwards towards the centre. The lens increases slowly in size during life by the superimposition of new fibres derived from the anterior endothelium. Any pathological change in the eye affecting the circulation must necessarily affect the lens. In old age nutritive changes are always seen. In the young adult, however, such changes are so frequently to be seen that I have searched for some cause which may be reasonably held to be responsible. I think this is to be found in glaucoma. In the case of glaucoma the circulation is interfered with to a great extent, and the functions of nutrition and excretion are much depressed.

In the first place there is diminution of the *quantity* of nourishment. This is brought about by wasting of the ciliary body consequent upon the compression of the filaments of the third nerve and trophic changes due to pressure on other nerves, and by the obstruction to the inflow of blood to all the internal parts of the eye. The *quality* of the nutrient media is lessened, owing to the inflammatory changes existing and the non elimination of waste products. Malnutrition, of course, directly affects the lens itself. The nuclear portion becomes smaller, and there is a certain amount of shrinking of the lens fibres. The stellate clefts are enlarged, and small vacuoles may be found. The cortical portion may be separated from the nuclear, the interspace being filled with fluid causing more or less opacity; or the endothelium may atrophy and become slightly opalescent.

In some cases of glaucoma one may see perfect examples of Morgagnian cataract, where the cortex of the lens has undergone liquefaction and the nuclear portion may be made to oscillate visibly in the capsule. This has been mistaken for true displacement of the lens. (*Vide* case below.)

Of the remote causes of glaucoma those which seem to me to be the most important are: (*a*) Heredity; (*b*) Inbreeding; (*c*) Distemper. Of these I only wish to refer to (*b*) Inbreeding. Those breeds of dogs which have for generations been inbred for fancy or show purposes seem particularly susceptible to eye troubles, and especially to glaucoma. The result of the glaucoma in these cases is early seen in the retina, which rapidly commences to undergo pigmentary degeneration. Pigmentary retinitis is seen in human beings as a result of marriages of consanguinity, and often affects several members of one family. The same remark applies to dogs, the disease running a protracted course and being quite intractable. The appearance of the retina is easily recognised with the aid of the ophthalmoscope. The pigment is laid down irregularly, and appears as black blotches on the surface of the retina; it seems to follow the course of the vessels, and sometimes to obliterate them. The patient nearly always shows marked photophobia in the earlier stages, and there is evidently a certain amount of pain. Glaucoma is always present, and very frequently the cornea shows some opacity. The condition, though incurable, may perhaps be arrested by sclerotomy and the frequent use of eserine.

To sum up the result of my observations—glaucoma is far more common in dogs than has been hitherto supposed. Unless checked early by operation it causes dimness of the lens, and in inbred dogs frequently gives rise to pigmentary retinitis.

I quote one typical case (from notes made at the time) showing all three conditions to a marked degree.

22nd Dec. 1903.—Brindle skye terrier with black points, three and a half years old, suffering from glaucoma and keratitis in the right eye, complete blindness, and apparent dislocation of the lens in the left. On the 29th, after treatment with lotions, the cloudiness in the right eye was less marked, but still only an imperfect view could be obtained of the interior of the eye. The dog is totally blind in the left eye, and has only partial vision in the right. He can see nothing directly in front of him, and has only a very imperfect vision of objects to the right of him. He guides himself entirely by the senses of sound and smell. On 9th January 1904, the eyes were very carefully examined with the aid of the ophthalmoscope. The left eye shows apparently complete dislocation of the lens, which is practically floating, and can be made to oscillate by gently swaying the dog's head.[1] The dog is totally blind in this eye. The right eye shows well developed glaucoma, the iris being pressed forward almost against the cornea. The lens is displaced upwards and outwards, and shows partial cataract (stellate). Pigmentary retinitis is present in both eyes, and is similar to the pigmentary retinitis of human beings.

This patient is very much inbred (his dam having been mated with her sire), and comes of an already inbred variety. As a puppy he had enjoyed very good health and apparently good sight, and at the present day he is a strong healthy dog, with a good coat, good bone, and straight joints, and in no sense "weedy." The glaucoma no doubt troubles him, and he suffers some discomfort from it making him very depressed. He is highly nervous, and his senses of smell and hearing are very acute—no doubt compensatory. The keratitis is consequent on the glaucoma. I can only account for the displacement of the lens in the right eye by interference with the function of the third nerve.

The prognosis in this case is not hopeful, the sight being almost certain to be entirely lost. Treatment consists of soothing and astringent lotions when there is evident pain, and $\frac{1}{800}$ gr. eserine night and morning to relieve ocular tension.

If no benefit is derived from this treatment I intend to perform sclerotomy.

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[1] This was evidently an error of diagnosis. The condition was Morgagnian cataract.

BILIARY FEVER IN THE HORSE.

By ALEXANDER EDINGTON, M.D., F.R.S.E., Director of the Government Bacteriological Institute, Grahamstown, Cape Colony.

THE malady in horses to which the above name is commonly applied in the Cape Colony would seem to have existed for very many years. As my duties necessitate my presence in the laboratory, I am compelled to fall back, for a description, on what has been stated in regard to it by various veterinary surgeons. At the same time, I may state that, in the course of inoculations which I have made into horses, conditions have been produced which are not unlike those described as pertaining to biliary fever. Before, however, describing these experiments, it may be of service to quote the published observations made by veterinary surgeons in the field.

In 1883 my colleague, Mr D. Hutcheon, the Colonial Veterinary Surgeon, described it in the following terms: "The disease to which I refer is very clearly described by Mr Wiltshire, Colonial Veterinary Surgeon for Natal, in his Report on Anthrax in the Natal Almanack for 1883. He terms it anthrax fever. Now, although I am inclined to differ in opinion with Mr Wiltshire respecting the nature of the disease, yet the symptoms of it as described by him correspond exactly with symptoms of the disease attributed to bots in this Colony. The following are the symptoms of the disease to which I refer. At first the horse appears dull, lacking his ordinary energy, and when feeding manifests a disrelish for his food."

Mr Hutcheon then refers to the fact that the lining membrane of the eye-lids, the conjunctiva, is of a dull brownish colour, "marked with a few dark spots or streaks," which, as Mr Hutcheon remarked, "often get larger, and in bad cases give a deep claret colour to the whole conjunctival membrane." He then states that the animal gets drowsy, stands or walks with head depressed, and eats or picks in a listless manner. He may, however, "appear uneasy, paws with his forefeet, bites at his sides, rubs himself against other horses, and manifests symptoms of abdominal irritation. This is succeeded by delirium. The animal will wander about the veldt in a heedless and stupid manner . . . the chances are that he will tumble over some krantz or into a deep sluit.

"In most cases the action of the bowels is arrested . . . It would appear, however, that constipation is by no means a constant symptom of the disease." Mr Hutcheon states "that diarrhœa was a constant accompaniment of it in 1879. The urine is generally high-coloured; in one case that I saw it was as dark as claret."

Mr Hutcheon, in describing the *post-mortem* appearances, states that the symptoms may vary a little, "depending a good deal on whether the horse has suffered from any previous disease." He then mentions the following as among the conditions found: "The abdomen generally contains a variable quantity of a brownish fluid; liver enlarged and congested . . . The spleen is generally enlarged and somewhat pulpy. The mucous lining of the upper portion of the small intestine is often congested and contains a quantity of viscid mucus . . . A small quantity of a straw-coloured fluid is generally found in the chest and heart bag; the lungs are more or less congested, and the interlobular tissue contains a variable quantity of a yellow, jelly-looking serum, *exactly similar to what is found in great abundance* in the real horse-sickness."

Dr Theiler, in the *Transvaal Agricultural Journal* for April 1903, in the course of an article on biliary fever, emphasises the observation of the yellow staining of all visible mucous membranes (jaundice), and adds that this continues for a considerable time and only disappears gradually. "The animal's temperature rises considerably, and is then accompanied by a very marked depression. Head and ears hang down and the eyes close. At this stage the symptoms may increase very suddenly and become alarming in their intensity. Death ensues, preceded by coma."

Among the conditions found on *post-mortem* examination are, "a brownish-yellow discoloration of the flesh . . . The liver is increased in size and shows discoloration. The spleen, with very few exceptions, is considerably enlarged . . . The pulp is dark brown in colour, and usually softened. All the lymphatic glands are more or less enlarged and of an œdematous character. The kidneys are pale and often enlarged. The lungs are pale and with a few exceptions normal. The heart is usually distinctly attacked, its flesh having a boiled appearance and very friable on the outside and inside lining; blood spots and blood streaks may frequently be observed. The heart ventricles are full of coagulated blood."

In the description by Mr Hutcheon it is stated that a serous exudation is found in the chest cavity and in the heart bag, and that a jelly-like exudation is found in the interlobular tissue. For the purpose of differentiation these facts are of the utmost importance, but in Dr Theiler's description they do not occur.

He states that he has failed to produce the disease by inoculating sound animals, and that doses of 50 to 100 cc. injected into the jugular vein, has always failed to produce any effect. He argues that either it only occurs in imported animals or the parasite is obliged "to pass through a phase of development outside the body of the horse, in the insect, before it is able to infect a fresh animal."

In the Report by Colonel Joshua Nunn, A.V.D., now Deputy-Director-General of the Imperial Veterinary Department, dated 16th October 1888, he states: "Four forms of horse-sickness have come under my observation, viz.: the 'acute' or 'pulmonary'; 'blue tongue'; 'dikkop,' which is the Dutch for a big or swollen head, and the 'subacute,' or what Mr Rutherford calls the bilious form."

After describing the former three, he states: "The subacute or bilious form comes on gradually, the animal being off his feed . . . The bowels are constipated, and fæces coated with mucus. The urine is often of a deep amber colour, the temperature of the body from 103 to 105° F., and the pulse quick and hard." He alludes to there being frequently abdominal pains present and sometimes violent and fetid diarrhœa. He also alludes to the presence of jaundice, and adds that cerebral symptoms may be present, and, if the latter, that the case is hopeless. He directs attention to the fact that "œdematous swellings appear on the body and limbs, which contain a quantity of yellow serum." He regards such swellings as of evil omen, and states that such cases usually terminate fatally.

In my Annual Reports for 1901 and 1902, I have given the details of a long investigation made by me into this subject, and I now propose, in alluding to these, to bring forward additional observations and conclusions which have been made during the year.

(1.) I have produced the disease by inoculation in more than forty horses. This fact abundantly testifies to the fact that the disease can be transmitted by inoculation.

(2.) I have produced it in animals born and bred in this district, thus showing that local horses are liable to the malady.

(3.) The majority of the deaths in the "protection camps" of this area occurred among animals from the Karroo. This suggests that Karoo animals are more liable to infection than coast-bred animals.

(4.) I have found that coast-bred animals are more or less protected.

(5.) The evidence I propose to bring forward will serve to show that many, if not most, animals running near the coast owe their seeming insusceptibility to the fact that they are already infected.

My first experience with this disease arose when I inoculated locally bred animals with the blood of a donkey that had been inoculated with horse-sickness blood ten days previously. Since then my former reports showed that the disease has been readily produced in clean horses by the inoculation of the blood of "salted" horses which had been inoculated with horse-sickness blood ten days previously. At a later date I found that the blood of the same "salted" horse would still produce the fever when drawn one hundred days after inoculation with horse-sickness blood.

Still later I determined that the blood of an "unsalted" horse could induce fever if the animal had recently been inoculated with the blood of the salted horse. In this case, however, the fever produced was not so severe as that obtained from a "salted" horse that had been recently reinoculated.

I now desired to determine whether the blood of sound but "unsalted" animals, which had been running in the veldt of this district for some time, could convey infection. For this purpose I borrowed a horse which had been running in the veldt at night and working during the day. By means of a hypodermic needle some of the blood was drawn from the jugular vein into a bottle containing a very small quantity of sterile neutral citrate of potash. Twenty cubic centimetres of this blood were injected subcutaneously into two clean horses, which I had received from the military authorities, and which had been under observation for several weeks after their arrival, previous to this inoculation.

In both animals about twenty-four hours after the temperature had risen I found parasites in the blood, and they continued in varying degrees to be present during all the febrile stages.

This experiment, therefore, fully justifies me in concluding that horses running in these areas are more or less generally infected with this disease.

As a corollary to the foregoing, and also by way of determining if I could raise the virulence, the following experiment was undertaken.

I borrowed another horse in Grahamstown, and in the same manner as before drew some blood. Forty cc. of this was injected subcutaneously into a clean horse (No. 3) on the 23rd December. A rise of temperature was seen on the seventh day. On the eighth day after the first inoculation 20 cc. of the same blood were injected subcutaneously and a similar amount intravenously.

On the 5th and 6th January, being the fourteenth and fifteenth days after the inoculation of horse No. 3, I bled it, and with the blood inoculated horse No. 4 by injecting 20 cc. intravenously on each day. Some slight fever was observed on the third day, which gradually increased, with very little remission. On the tenth day a further inoculation was made by injecting 20 cc. of the fresh blood of horse No. 3 subcutaneously. On the following day the temperature fell, but rose again on the twelfth day. The animal died on the thirteenth day. During the period when the temperature was highest there was a distinct hæmaturia, the urine being of a deep port-wine colour.

On making a *post-mortem* examination immediately after death I found the following conditions: As the animal lay on the table with the head and neck hanging down, a small amount of yellow gelatinous serous exudation ran from the nose. This material most evidently came from the trachea, in which some also was found. On dissecting the skin over the neck a small amount of gelatinous œdema was seen to lie around and to follow the course of the great vessels in the neck. On opening the chest almost half a gallon of dark yellow serous exudation was found. Both lungs were slightly congested, and some yellow gelatinous exudation was found under the pleural membrane covering the lungs and also in the interlobular tissue. The interlobular exudation was best seen in the left lung, and particularly towards the free edges. Along the posterior aorta a considerable amount of gelatinous œdema was found, and numerous petechiæ were seen lying somewhat superficially. About four ounces of yellow serous exudation was found in the pericardium. The outside of the heart showed numerous petechiae over the left ventricle and auricle, while in a lesser degree they were found over the right side. The base of the heart over the right ventricle was occupied by a large gelatinous œdema. The interventricular groove was occupied by the same œdema, while the blood vessels in this situation were infiltrated in the outer coat, appearing as white opaque lines. The spleen was enlarged, somewhat softer than normal on section, and covered with discrete petechiæ. The kidneys were pale and contained a small amount of gelatinous œdema in the pelvis. The liver looked as if boiled. There was evidence of old standing disease in the form of chronic venous congestion, while small discrete areas of necrosis were found throughout the organ.

Taking the *post-mortem* examination as a whole, the resemblance to horse-sickness was exceedingly great. The œdema of the pelvis of the kidney and the condition of the spleen are somewhat in excess of what one usually finds in horse-sickness, yet in some cases of that disease I have found in my experiments conditions exactly similar.

In my Report for 1898 I showed that when a dose of serum, taken from a horse which had recovered from horse-sickness and been subsequently reinoculated on several occasions, was given to a horse in which the fever of horse-sickness was just beginning to appear or had already set in, a hæmaturia appeared about forty-eight hours later.

After such a condition had appeared the animal died in less than forty-eight hours. The *post-mortem* examinations in these cases were absolutely identical with that just described.

The serum then used was prepared in the following manner. Horses which had been "salted" to horse-sickness were reinoculated periodically with increasing doses of virulent horse-sickness blood until doses of 200 cc. could be given without producing any evil effect. Some little time thereafter these animals were bled, and the serum was then filtered through a Berkefeld filter (which keeps back all organisms which are visible under the microscope), and thereafter 0·5 per cent of carbolic acid was added to it and the whole filtered again. The serum was frequently used some weeks after having been made, and this fact, taken in conjunction with the mode of preparation, completely precludes the idea that it could have been the means of conveying a gross malarial infection.

The interest in the case, however, lay in the remarkable fact that the serum possessed no power to produce harm in healthy animals. When given, however, to animals suffering from horse-sickness, it produced the remarkable hæmolysis to which I have alluded.

It is noteworthy that Dr Koch found the serum from recovered Rhodesian redwater cattle to possess this same remarkable property.

As already stated, I considered the condition I produced experimentally as a malarial form of horse-sickness, and that it agrees with Colonel Nunn's description of the "bilious form of horse-sickness."

In the cases where I produced it by inoculating with the blood of healthy horses, I considered it as identical with the so-called biliary fever. When these latter animals recovered they were then inoculated with the blood of a recently reinoculated "salted" horse. Since no fever resulted therefrom, I am disposed to consider the conditions produced as identical, and that the condition denominated by me as the malarial form of horse-sickness is the same as that known as biliary fever.[1]

In my Report for 1902 details of experiments are given which strongly support my view that the malarial condition is one phase of horse-sickness. If, however, a critical investigation were required to prove the matter absolutely, it would be necessary to carry these out in a country where the natural infection does not obtain, so as to eliminate any fallacy from any occult malaria.

Protective Inoculation against so-called Biliary Fever.

During my experiments I have attempted the production of the fever in fifty horses, of which the results may be summarised as follows:—

(1.) Sixteen proved absolutely insusceptible.

(2.) Twenty-four showed high fever, and were thereafter insusceptible.

(3.) Three showed slight fever, and were thereafter insusceptible.

(4.) Three showed slight fever, and after a second inoculation the fever increased.

(5.) Two showed no fever until the second inoculation.

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[1] "Journal of Hygiene," Vol. IV., No. 1, 1904

(6.) Two showed slight fever, which was continued after the second inoculation.

From the above, therefore, it is seen that as a rule one attack renders the animal thereafter insusceptible.

Where second attacks are seen to have arisen in the foregoing experiments, it deserves to be noted that the second inoculations were made within a month of the first, and that experience has shown that the malarial fever is not always completely ended by that time.

Under such circumstances, and seeing that, as a rule, no serious illness follows the inoculated disease, I can confidently recommend inoculation to be practised in such areas as the disease commonly occurs in. For this purpose I propose to supply blood drawn from properly prepared animals and at a moderate cost. I would recommend a dose of twenty cubic centimetres to be injected subcutaneously, and the animal thereafter permitted to rest for a month before being worked.

Farmers should note that the blood must be used as soon as possible, and that the animal must be stabled during its month's rest.

IMPLANTATION OF TISSUE AND ITS RELATION TO CANCER.[1]

By EDWARD H. NICHOLS, M.D., (Assisting Visiting Surgeon, the Boston City Hospital; Assistant Professor of Surgical Pathology, the Harvard Medical School.) (From the Surgical Laboratory of the Harvard Medical School.)

BELOW are presented the results of a series of implantations of tissue in sixty-two animals. For implantation both adult and fœtal tissues were used. The implanted tissues were chiefly epithelial, although some mesenchymal tissues were used. The experiments were undertaken to see if epithelial or mesenchymal tissues, set free from their normal environment, could acquire a power of unlimited growth and give rise to the formation of metastases. In some cases the implanted tissues proliferated, maintained their vitality, and produced nodules analogous to dermoid cysts or teratomata, and some of the nodules were of large size. In no case was any nodule produced with unlimited growth or the formation of local or glandular metastases.

This line of experimentation was undertaken because, from a previous study of the inclusions in cancer cells, from experiments with the so-called "cancer yeasts," from a study of various lesions claimed to be due to protozoic infection, and from a study of the literature on the infectiousness of cancer, it was believed that there was less evidence that cancer was due to a parasite than that it was due to some inherent character of the epithelial cells. A critical review of the reasons which led to this belief follows.

The investigators of the subject of the origin of cancer and other

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[1] Reprinted from the "Journal of Medical Research" Jan. 1905.

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malignant tumours may be divided into two classes. One class believes that cancer is due to a living parasite, the other that the epithelial proliferation in cancer is due to some biological peculiarity of the cells themselves. By those who believe in the parasitic nature of cancer certain general reasons are advanced to support the general assumption. The men who accept the parasitic theory of origin may be divided into two classes, some believing that the parasite is a blastomyces, others that it is a protozoon. By those who believe that the development of cancer is due to biological causes, various hypothesis, supported by various known clinical and experimental facts, have been advanced. The hypothesis which have found widest acceptance are those of Thiersch, Cohnheim, and Ribbert, while with these three should be mentioned the theory recently advanced by Bashford and Murray. The evidence for and against all of these ideas will be taken up in the order in which they have been mentioned.

I. GENERAL ARGUMENT FOR THE PARASITIC NATURE OF CANCER.

1. *Apparent Relative Increase in Frequency of Cancer.*

If a relative increase in the number of cases of cancer could be demonstrated, it would tend to show that the disease was infectious. It is claimed that there is such a relative increase, and that the increase is general in all countries; that there is also a local increase in certain regions, and that certain houses are especially associated with the occurrence of cases of cancer. The statements are based upon a study of statistics. The inherent weakness of statistics is too evident to require comment. It is noteworthy that statistics from men who favour the parasitic theory are invariably in favour of their contention. The relative value of statistics in support of such a theory must in the nature of things be of small, and if not verified by other facts, of almost no value. Without going into a detailed comparison of the various published statistics, it may be said that the result of statistical evidence is about as follows:

In earlier days the term "cancer" included many lesions the cause of which is now known. This removes such lesions at present from the class of cancers. This fact *per se* should lessen the apparent number of cancers. On the other hand, from improved clinical diagnosis, and from the frequent occurrence of pathological and histological examination, many deaths, formerly obscure, are now classified as due to cancer. Moreover, an improvement in the requirements regarding the registration of the causes of death by many countries has led to an increase in the number of cases of cancer reported. It has been shown[1] that the relative number of cancers is greater in cities than in rural districts, probably because of better registration and the greater likelihood that the cases will be seen by physicians. There also has been an increase in longevity on account of the lessened death rate from acute infectious diseases. Since cancer notably is a disease of later life, this would explain some of the apparent increase in frequency. Finally, as Newsholm[1] remarks, the statistics as to the cause of death represent the clinical opinion of medical men and not the facts, *e.g.*, Bollinger[2] shows that of seven hundred and eleven cases of cancer seen at autopsy at the Friedrichshain Hospital, in twenty-two per cent. no diagnosis of cancer had been made during life. Newsholm believes that the increase in cancer is only an apparent one, due to improved diagnosis and better registration, but that absolute proof on either side is impossible because of the imperfect data available. He believes that there has been no real increase during the last forty years.

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[1] Bollinger. Die Haufigkeit des Karzinoms in München. "Münch. Med. Wochenschr.," 1903, No. 38

Statistical evidence of local frequency.—It has been claimed from a study of statistics that certain local causes favour the occurrence of cancer, and this fact has been considered to show that cancer is infectious. Wolff[3] *e.g.*, showed that the distribution did not depend upon geographical conformation, rainfall, or elevation, but tha cancer was apparently more frequent in great river valleys and in wooded districts. Bashford and Murray[4] have shown, however, that cancer occurs in all of the domestic animals and in various wild animals, including mammals, birds, reptiles, and fishes. This certainly suggests that, on account of the varying conditions of life and environment under which these different animals live, some general cause must be found, and that in all probability no local cause can be operative. Bashford and Murray also show that cancer occurs in all countries, although it is apparently less frequent in the less civilised ones, and believe that this is due to less careful observation.

It also has been claimed by many that cancer occurs more frequently in certain local districts, and, [5] [6] [7] *et al*, more especially, in certain houses. On this subject again there is a dispute as to facts. The wide zoological distribution of the disease as just mentioned would make such a local increase theoretically unlikely, and Newsholm says that "observations of considerable magnitude, making all necessary corrections for age distribution of the population, would be required to give a *prima facie* case to establish a high degree of probability for the contention that there are special 'cancer houses,' and such observations as have hitherto been published do not carry conviction to my mind."

On the whole, it may properly be said that statistics *per se* must be considered a weak support for the theory of the infectiousness of cancer; that there is a dispute as to the accuracy of the facts and to the conclusions which may be drawn from them; that there is no positive evidence that there has been a relative increase in the number of cases of cancer not explicable on other grounds than infectiousness, and that there is very good evidence that local causes are not active in the causation of the disease.

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[1] Newsholm. The Statistics of Cancer. "The Practitioner," 1899, p. 371.

[2] Bollinger. Loc. cit.

[3] Wolff. A comparative study of cancer mortality. "Brit. Med. Jour.," 1903, p. 929.

[4] Bashford and Murray. The zoological distribution of malignant new growths. "Sci. Rep. Cancer Research Fund," No. 1, London, 1904.

[5] Lyons. A statistical study of a rural cancer district in the state of New York. "Fourth Ann. Rep. Cancer Lab., N. Y. State Board of Health," p. 97.

[6] D'Arcy Power. A further contribution to the distribution of cancer. "Practitioner," May, 1903.

[7] Behla. Ueber die Krebserkrankungen in Luckaü. "Zeitschr. f. Med.," 1901.

2. *Formation of Metastases by Cancers and other Malignant Tumours.*

In many of the infectious diseases due to a parasite, *e.g.*, to bacteria, by distribution of the parasite through the blood or lymph vessels, secondary foci are produced in parts of the body at a distance from the original point of infection. It also is well known that a primary nodule of cancer or sarcoma may be followed by secondary nodules in various parts of the body through extension. This occurrence of secondary nodules in cancer has been claimed to be analogous to the metastases in known parasitic diseases, and has been claimed to indicate the infectiousness and parasitic origin of cancer.

In fact the character in the metastases in cancer is probably the most convincing theoretical argument against the parasitic nature of the disease. Identity of method of extension of two diseases is no proof of identity of origin. It has been shown,[1] *e.g.* that normal tissue introduced into the circulation may be carried to various parts of the body at a distance from the original point of inoculation, may lodge and grow. In bacterial diseases the primary area of reaction is due to reaction of the fixed tissues to the irritation produced by the bacteria. The secondary areas, or the metastases, are produced by the reaction of the fixed tissues of that part of the body to which the bacteria or irritant is carried, but do not represent a transfer of the cells of the primary nodule. In cancer the facts are very different. The secondary nodules always represent a transfer and proliferation of the cells from the primary nodule, and are not produced by the reaction of the fixed cells of the part, *e.g.* if, with an epidermoid cancer of the lip, metastases are formed in the liver, the metastases are always identical histologically with the primary nodule. If the disease were due to a parasite which had the power of producing an unlimited proliferation of epithelial cells, the secondary nodule in the liver should be composed of cells resembling the liver epithelium. Such never is the case, the histological type of the original nodule is invariably reproduced. To explain this reproduction of the original type on a parasitic basis, it would be necessary, as Ribbert has shown, to assume that the parasite which produces a given type of epithelial proliferation of cancer must be specific for that type of epithelium only, that it must always live within those cells, and must divide at the same rate as the type of cell within which it lives. Since cancers occur resembling all types of human epithelium, this would require just as many types of specific cancer parasites as there are types of human epithelium. Moreover, since it has been shown that cancer is widely distributed in the animal kingdom, and since, as will be shown later there is no proof that cancer of one species of animal can be transferred to an animal of another species, just as many specific parasites would be required as there are types of epithelium in all of the various species of animals in which cancer occurs. *A priori* such a series of parasites seems improbable at least. Moreover, this assumption does not agree with the statements regarding the occurrence in the tissues of various alleged cancer parasites. Roncali, *e.g.*, claimed to produce an adeno-carcinoma, an epidermoid cancer, and a sarcoma with one and the same parasites.

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[1] Cohnheim and Maas. Zur Theorie der Geschwulstmetastasen. "Virch. Arch.," LXX.

Gaylord[1] claims to have produced epithelial proliferation in the lung by injection of ascitic fluid from abdominal cancer, and to have had cancer of the liver follow inoculation with ascitic fluid from an abdominal cancer. He also claimed to find the "parasite" in the circulation, in effusions, and in the tissues. Von Leyden[2] claims to have found the parasite outside the cancer cells, and in a previous report by the Cancer Committee of the Harvard Medical School the bodies claimed to be parasites have been seen outside cancer cells.

In this connection it should be borne in mind that Locke[3] showed that the growth of cancer is by direct extension of the cancer cells from one focus, and not by extension from various foci. Petersen[4] after similar study, came to the same conclusion. This fact would argue against a proliferation of epithelial cells produced by a parasite disseminated outside of the cells, attacking several scattered areas at approximately the same time.

3. *Occurrence of Cachexia in Cancer.*

In patients suffering with cancer there occurs a peculiar cachexia. which often appears to be out of all proportion to the size of the tumour. Since in some diseases known to be due to a parasite a similar cachexia exists, it has been assumed that the cachexia in cancer also indicated the presence of a parasite. Clinically, however, the amount of cancer cachexia is very variable; it may be absent or marked with a small cancer, or slight with a large one. There is much clinical evidence to show that the amount of cachexia is dependent upon the site of the cancer; it is greatest in cancers which ulcerate early or are so situated as to give a chance for secondary bacterial infection, rather than due to the primary effect of an alleged parasite.

4. *Possibility of Direct Transfer of Cancer from Person to Person.*

It is claimed that it is possible to transfer cancer from a person suffering with the disease to a well person by direct contact or inoculation. Behla[5] reports seventeen cases, chiefly of his own observation, of what he considers transfer of this sort, and believes that these cases argue the infectiousness of cancer. An analysis of his cases shows that in but a small number was the affected epithelium or the cancer in the two individuals of the same type. In most cases the type of cancer was different in the two individuals, *e.g.*, cancer of the stomach in one, and epidermoid cancer of the lip in the other, etc. Bearing in mind the nature of cancer metastases, already mentioned, the infection in these cases does not seem proven. Boas[6] reports five cases in which both husband and wife had cancer of the stomach. Elsler[7] reports a case of cancer "à trois"—the first individual having a cancer of the rectum, the second cancer of the lip, and the third cancer of the rectum. Guermonprez[1] reports the case of a surgeon who examined a woman with cancer of the uterus while he had an acne pustule on the finger. Afterwards the surgeon had a cancer of the finger. Guermonprez himself operated on a cancer of the face and injured his finger. A nodule afterwards appeared on the finger which resisted the cautery for nine months.

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[1] Gaylord. On the presence of a characteristic organism in cancer. "Third Ann. Rep. N. Y. State Path. Lab.," Buffalo, 1901.

[2] Von Leyden. Zur Ætiologie des Carcinoms. "Zeitschr. f. Klin. Med.," XLIII., p. 1, 1901.

[3] Locke: The reconstruction in wax of a nodule of cancer. "First Ann. Rep. Cancer Committee, Harv. Med. Sch., Boston," 1900, p. 39; "Journ. Bost. Soc. Med. Sciences," V.

[4] Petersen: Ueber Aufbau, Wachstum, und Histogenese der Hautcarcinome. "Beiträge z. clin. Chir.," 1902, XXXII., p. 3.

[5] Behla: Uber "Cancer à deux." "Dtsch. Med. Wochensch.," XXVI., 1901.

[6] Boas: Statist-Klinisch. Studien an 200 Fällen von Krebs des Magen-Darmkanal. Verhandl. des XVIII. "Congr. f. inn. Med. zu Wiesbaden," 1900, p. 374.

[7] Elsler, quoted by Behla. Loc. cit.

Budd[2] reports a nurse who washed the linen of a woman with cancer of the uterus, who afterwards had cancer (sic!) of the axilla. The cases can be indefinitely multiplied, but the character of the evidence can be judged from the examples given. These cases can be divided into four groups, where people living together in close association are affected by (1) cancer of the same type where direct contact is possible, *e.g.*, cancer of uterus in wife, with epidermoïd cancer of penis in the husband; (2) cancer of the same type or organ where direct contact is not possible, *e.g.*, husband and wife each having cancer of the stomach; (3) cancer of different types where contact is possible, *e.g.*, cancer of the breast in one and cancer of hand in the other, and (4) cancer of different types where no contact is possible, *e.g.*, cancer of stomach in one and cancer of liver in another. The first class, which is the most suggestive of all, is extremely rare, and when one considers the frequency with which, *e.g.*, both uterus and penis are affected by epidermoid cancer, it is obvious that such cases are quite as readily explained by coincidence as by infection. Moreover, considering the latency of many cancers of the uterus, and the fact that unsuspected contact cannot be rare, the evidence of infection is slight. In the second class coincidence again seems as justifiable an explanation as infection, especially in view of the rarity of the cases. As evidence in so complicated a subject as cancer such cases at best seem only suggestive, and, as proof, are too trivial in view of other evidence. In the third and fourth classes the improbability of infection in view of what has been said in regard to cancer metastases is very great. On the whole, it can be said that evidence of the transfer of cancer by contact is slight, and proof is absolutely lacking. Foulerton,[3] in a discussion of this subject, says that "there is no evidence worth considering that there is such a thing as direct personal infection."

5. *Self-inoculation by Cancer.*

The transfer of cancer from one part of the same individual to another by transplantation has been demonstrated. Cornil[4] reports two cases which were told to him by the surgeon who did the transplanting. In one case a woman had a fibro-sarcoma of the breast, which was removed by operation. A bit of the tumour was buried in the second breast, and a tumour developed at the point of inoculation with the same histological structure as that of the original tumour. with many mitoses present. In the second case a woman had a tubular cancer of one breast, which was removed by operation, while a bit was implanted in the sound breast. After some months a tumour with structure similar to that of the original tumour developed at the point of inoculation. Cornil expressed his great disapproval of the experiments. Hahn[1] transplanted a bit of cancerous skin to another part of the same individual and witnessed a cancer of the original type develop at the point of inoculation. Moreover, similar implantations have occurred accidentally during operations upon cancerous tumour. The recurrence of malignant tumours in the scar of the operation at some distance from the original location is well known, and frequently has been reported. Richardson[2] reports an infection of the pectoral muscle with cancer by the use of a trocar to remove a bit of tissue from a breast tumour for histological examination. Implantation of cancer by contact in the same individual also occurs with reproduction of a tumour of the original type, *e.g.*, epidermoid cancer of the lower lip may cause a cancer of the upper lip if the two are in contact. The same thing is true of the female genitals, etc. Ebert[3] reports twenty-two such cases, but believes that they all can be explained by accidental implantation, and that they do not represent an infection of adjacent epithelium by a parasite, and therefore cannot be used as an argument for the infectiousness of cancer. In all of these cases it is to be remembered that the original type of epithelium always is reproduced.

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[1] Guermonprez: De la contagion professionnelle du cancer. "Bull. Méd.," 1896, p. 251.

[2] Budd: Is cancer contagious? "Lancet," II., 1887, p. 1091.

[3] Foulerton: The causation of carcinoma and sarcoma. "Practitioner," 1902, July, August.

[4] Cornil: Sur les greffes et inoculations de cancer. "Bull. de l'Acad. de Méd.," 1891, No. XXV.

6. *Direct Inoculation of Human Cancer into Animals.*

There are many alleged cases of the direct transfer of human cancer to animals, and this fact has been used as evidence of the contagiousness of cancer. Langenbeck,[4] in 1840, injected the juice of a medullary cancer of the humerus (sic !) mixed with defibrinated blood into the femoral vein of a dog. The animal died in two months, and showed nodules in the lungs which histologically were "carcinomatous tubercles." There are no drawings, and the description of the histology makes it probable that the nodules were probably areas of granulation tissues and giant cells about cancer cells acting as foreign bodies. There is no evidence of proliferation of the transplanted cancer cells or of true tumour growth. Follin[5] injected into the jugular vein of a dog an emulsion from a cancer of the breast. The animal died in fifteen days, and showed nodules on the walls of the heart and in the liver in which "cancer cells" could be seen. Here again there is no evidence of growth of the cancer cells, which probably acted as foreign body emboli. Lanz[6] inoculated guinea-pigs with an emulsion of a melanotic sarcoma, and in one animal found many pigmented cells, but did not produce a tumour of the original histological type. Francotte and de Richter[7] inoculated ten white rats subcutaneously in the breast with cancer juice. Each animal was inoculated with juice from seven different tumours of varying histological structure, epidermoid, scirrhus, and encephaloid, at different intervals, extending over a period of six months. No result was obtained until three months after the last injection. Then at varying intervals a nodule of new tissue appeared in each of the animals near the point of inoculation, which in most cases led to necrosis and spontaneous amputation of the adjacent limb. After this amputation the animals usually recovered their general health. The observers studied the tissues in two of the animals. In one case they found an epidermoid cancer, with similar metastases in the adjacent lymph nodes. In another case they believed they saw an alveolar cancer of the breast, without metastases in lymph nodes. The paper was presented as a preliminary report, and the writers were to study the nodules in the remaining animals. No report has yet appeared, so that it is fair to presume that in the other cases no cancer was found. Transplantations to other animals were attempted. There is no proof that the cancers came from the inoculated cancer juice. Mayet[1] inoculated rats with a glycerine emulsion of human cancer tissue, and in one animal found a cancer nodule in the kidney. There is no evidence that this nodule was the result of the inoculation. Boinet[2] inoculated sixty animals with human cancer tissue. All subcutaneous inoculations with masses of tissue failed. In one case a rat inoculated in the pleura with the juice of a lymphosarcoma showed a nodule of cancer (sic!). He also inoculated a rat peritoneally with tissue from a cancer of the penis, and after two months found two cancer masses the size of a cherry below the diaphragm, attacking the vertebræ, compressing the cord, and causing paralysis. Here again is no evidence that the tumour was derived from the implanted tissue. Bosc and Vedel[3] inoculated rabbits with human cancer tissue, and got at the point of inoculation "new formations," adherent to the skin with enlarged adjacent lymph nodes. Where the nodule was adherent to the skin there was a proliferation of the Malpighian layer, and many "cancer bodies" were seen. By similar peritoneal inoculations they got nodules in the liver and spleen composed of "connective tissue and large nucleated cells." They inoculated "guinea-pigs with the tissue of a melanotic sarcoma subcutaneously and intraperitoneally, and obtained nodules "with the structure of melanotic sarcoma." They also inoculated dogs peritoneally. In one case they put tissue from a cancer of the lip into the peritoneal cavity, and after two months found a nodule like that of the lip in process of evolution. In another case, after inoculation with tissue from a cancer of the breast, they found a large nodule the size of an orange; a bit of this nodule in the second dog was followed by the formation of a large nodule, and a piece from this second nodule in still a third dog showed disseminated nodules on the peritoneum. In this case there is no histological evidence of the cancerous nature of the nodules. They also inoculated a dog in the peritoneal cavity with a large piece of encephaloid cancer, and in two and one-half months found a nodule of "alveolar structure" with many cancer bodies. In none of Bosc and Vedel's cases is there any histological evidence of the correctness of their claims. Reale[4] transplanted a bit of skin sarcoma into a rabbit's subcutaneous tissue, and after two years a nodule was found which histologically was a lymph-angio-sarcoma, *i.e.*, not of the original type. Dagonet[1] inoculated the peritoneal cavity of a rat with tissue from a pavement-celled cancer of the penis. The animal died in eighteen months with gradual emaciation. Nodules were found in the omentum, spleen, and liver, but the peritoneum was normal. The nodules histologically were pavement-celled cancer. In this case there is no proof that the nodules came from the transplanted tissue. Many men have reported absolute failures in attempts to transplant human cancers to animals. I have made between twenty and thirty such attempts, using rabbits and guinea-pigs for animals, and transplanting human cancers, both of the epidermoid and glandular types, and transplanting tissue bits and emulsions both subcutaneously and peritoneally. There never resulted any tumour formation or growth of the transplanted cells. In some cases the nodule transplanted suppurated, in others it finally disappeared, leaving only scar tissue. Gaylord[2] inoculated guinea-pigs with peritoneal fluid from a case of human abdominal cancer, and found in the lung of one animal a small nodule which he considered to be adeno-carcinoma. In another similar case he inoculated a dog with peritoneal fluid and killed the animal after ninety days. He found numerous nodules in the liver which he considered new formations derived from the liver epithelium. There is no proof of the reproduction of the original type of cancer. Hansemann,[3] who had opportunity to study the tissue from both these cases, denied that the nodule in the guinea-pig was a true adeno-carcinoma, and stated that similar appearances could be seen in human lungs in chronic induration. He also declared that the nodules in the dog's liver were not primary nodules, but were nodules secondary to some overlooked primary focus.

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[1] Hahn: Ueber Transplantation carcinomatöser Haut. "Berlin Klin. Wochenschr.," 1888, XXV., p. 413.

[2] Richardson: "Bost. Med, and Surg. Jour.," CXXXIX., p. 414, 1898.

[3] Ebert: Zur Casuistik des Infectionstät des Carcinoms. "Inaug. Dissert.," Berlin, 1895.

[4] Laugenbeck: "Schmidt's Jahrb.," XXV., 1840.

[5] Follin: "In Lebert's Traité pratique des malades cancéreuses," Paris, 1851, p. 136.

[6] Lanz: "Kocher's Festschrift," Wiesbaden, 1891.

[7] Francotte and de Richter: Récherches experimentelles sur le cancer. "Bull. de l'Acad. Royale de Med. de Belgique," XLIV., p. 999, 1892.

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[1] **Mayet: "Sem. Méd.," 1893, p. 283.**

[2] **Boinet: Transmission aux animaux du cancer de l'homme. "Sem. Méd.," 1894, p. 213.**

[3] **Bosc and Vedel: "Sem. Méd.," 1898, p. 166; Presse Méd.," 1898, p. 213.**

[4] **Reale: "Tentativo d'inoculazione sperimentale del sarcoma sutaneo," Naples, 1902.**

Another fact which makes these reported successes theoretically improbable, is the fact, as will be shown later, that in some cases of cancer in rats where successful transplantation from one rat to another has been successfully and readily carried out, there has been absolute failure in all attempts to transfer the same tumour to animals of another species, *e.g.*, to guinea-pigs or rabbits.

On the whole, in regard to these experiments, it is fair to say that in the literature there is no proved or authentic case of the transfer of human cancer to another animal, and that the evidence in favour of the claim of such transference is in no case of such a character as to make the accuracy of the claim even probable.

7. *Inoculation of Cancer or Malignant Tumour into Another Animal of the Same Species.*

A considerable series of experiments showing the possibility of transferring cancer or other malignant tumours from one animal to another of the same species exists, and these results in several cases have been carefully verified by exact and painstaking work. Hanau[4] put tissue from an epidermoid cancer of a rat into the scrotum of another rat and got a similar epidermoid cancer at the point of inoculation, but had no metastases in the lymph nodes. Loeb[1] transplanted tissue from a cystic sarcoma of the thyroid of a rat to other rats, and produced at the point of inoculation a tumour of histological structure similar to that of the original tumour. These transplanted tumours infiltrated surrounding tissues, but did not form metastases. He could not inoculate the tumour into other species of animals. He showed that the new growth came from a proliferation of the transplanted cells and not from infection of the fixed cells of the inoculated animal, *i.e.*, that the new-formed tumour was a true transplantation and not an infection. He also successfully transplanted into other rats tissue from a mixed tumour of the thyroid of a rat. Inoculation of rats with juice from the tumours after filtration to remove tumour cells gave no growth. In neither case could organisms of any sort be cultivated. Velich[2] transplanted tissue from a periosteal sarcoma of the thigh of a white rat to other rats, and at the point of inoculation got tumours whose structure constantly was like that of the original tumour. After some generations of success the power of being transplanted was lost. Inoculations with tumour juice from which cells had been removed failed. It is noteworthy that uninoculated animals kept in the same cage with inoculated animals developed similar tumours. In his experimental animals no metastases were formed. Morau[3] inoculated rats with an emulsion of tissue of a cylinder-celled adenoma of the axilla of a rat, and produced at the point of inoculation tumours with the same histological structure. The transplantations were carried through several generations, but the inoculalibility of the tumour finally diminished. No organisms could be cultivated. Borrel[4] inoculated rats through six generations with an epidermoid cancer of another rat. Transplantations succeeded in about ten per cent. of the cases. In some of the inoculated animals metastases incurred with involvement of lymph nodes. The tumours appeared at the point of inoculation, and their histological structure was identical with that of the primary tumour. Rats kept with inoculated animals also developed cancers. Jensen,[5] in the most valuable contribution yet made to this subject, reports successful transplantation of skin cancer from a mouse to other mice. The inoculated animals developed tumours at the point of inoculation, the histological structure of which was identical with that of the original tumour. He carried successful implantations through nineteen generations, and succeeded in about one-third of the animals. He was unable successfully to transplant to other species of animals. He saw no parasites in his experimental tissues, nor was he able to cultivate any organisms. He inoculated rabbits with tumour tissue from rats, and from the rabbits obtained a serum which in rats destroyed the tissue of experimental tumours. He inoculated rats with tissue from their own tumours, and was inclined to believe that he thus obtained immunity in the animals. He also believed that with serum obtained from animals inoculated with tumour tissue, and so made actively immune, he could protect rats from transplantation (passive immunity). Young tumours were less successfully transplanted, and Jensen believes that this is due to the fact that the rapidly mitosing cells of young animals cannot stand interference with their nutrition as successfully as the less rapidly growing cells of old tumours. None of Jensen's tumours formed metastases. Jensen showed that the tumours arose from proliferation of the transplanted cells and not from infection of the fixed cells of the inoculated animal, *i e.*, that they were true transplantations. In a few animals endocellular inclusions were seen, but Jensen says that they were degenerations.

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[1] Dagonet: Transmissibilité du Cancer. "C. R. Soc. Biol.," IV., 1903.

[2] Gaylord.

[3] Hansemann: "Verhandl. des Comités f. Krebsforschung," 1902-3, Heft. II., p. 34.

[4] Hanau: Erfolgreiche experimentelle Ubertragung von Carcinom. "Fortschrit. des Med.," 1889, IX., p. 321.

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[1] Loeb: Transplantation of tumours. "Jour. Med. Research," VI., p. 28. Also Uber Transplantation von Tumoren. "Virch. Arch.," CLXXII., p. 172, 1903. Also Mixed tumours of thyroid gland. "Am. Jour. Med. Sci.," February, 1903, p. 243. Also Further investigations in transplantation of tumours. "Jour. Med. Research," VIII.

[2] Velich: Beitrag zur Frage nach der Ubertragung des Sarkomes. "Wien. Med. Wochenschr.," 1898, p. 711.

[3] Morau: Récherches exper. sur la transmissibilité des certains néoplasmes. "Arch. de Méd. expér. et d'Anat.," September 1894, p. 677.

[4] Borrel: Epithélioses infectieuses et epithéliomas. "Ann. de l'Inst. Pasteur," February 1903, p. 81.

[5] Jensen: Experimentelle Untersuchungen über Krebs bei Mäusen. "Centralbl. f. Bakt.," XXIV., 1903, p. 28.

These experiments show the certain possibility of the transplantation from one animal to another of the same species of tumour tissue, both cancer and sarcoma, with formation of tumours at the point of inoculation of histological structure identical with that of the primary tumour. This transplantation can be carried through many generations, although not every individual experiment succeeds. In very few instances endocellular structures are seen, due to degeneration. In no case has any organism been cultivated from the experimental tumours. All observers agree that the tumours are true transplantations and that the tumours develop from proliferation of the transplanted cells and not from infection of the fixed cells of the inoculated animals. When tumour cells are excluded by filtering, in no case does the injected cancer juice cause tumours. In but few cases were metastases or lymph node infection produced.

8. *Parasitic Production of Epithelial Proliferation Analogous to the Epithelial Proliferation in Cancer.*

In support of the parasitic origin of cancer it has been claimed that various other diseases characterised by marked proliferation of epithelium are due to the action of a protozoon parasite. The diseases mentioned in this connection are coccidiosis, Darier's disease, molluscum contagiosum, and a class of diseases which Borrel includes under the term "epithélioses infectieuses," *i.e.*, clavelée or sheep pox, variola, varioloid, etc. Tyzzer[1] has shown that the primary effect of the coccidium oviforme is a destruction of epithelium with a secondary formation of connective tissue, which later becomes covered by epithelium, and that the real lesion is of the nature of a chronic inflammation. Darier's disease has never been proved to be of parasitic origin, and that opinion now is generally discredited. As regards molluscum contagiosum, White and Robey[2] have shown that no parasite ever has been demonstrated in the disease. As regards the "epithélioses infectieuses," although in these diseases there is a marked proliferation of epithelium, the proliferation is not analogous to the proliferation of epithelium in cancer. Borrel[3] has shown that the secondary nodules in the lungs in clavelée are derived from the fixed epithelial cells of the lung, while, as has already been shown, the secondary metastases in cancer are of an entirely different origin. Borrel says that the morphology of the cell inclusions in clavelée, etc., is not like that in cancer. . Dr Councilman assures me that the intracellular bodies in variola and varioloid are not analogous with those of cancer.

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[1] Tyzzer: Coccidium infection of the rabbit's liver. "Second Ann. Rep. of the Cancer Committee, Harv. Med. School, Boston," February 1902, p. 1; "Journ. of Med. Research," VII.

[2] White and Robey: "Molluscum Contagiosum," same reference, p. 23.

[3] Borrel: Loc. cit., supra.

Summary of Conclusions from General Arguments for Parasitic Origin of Cancer.

From a consideration of the facts already presented it may be said that (1) the relative increase of cancer is disputed and not proved; (2) local causes of the origin of cancer are improbable; (3) the metastases in cancer, although taking place by the same routes as the metastases in infectious diseases, are of an absolutely different origin, and constitute the strongest theoretical argument against an infectious or parasitic origin; (4) the cachexia in cancer probably is not entirely due to the character of the cancer itself, but is often dependent upon secondary bacterial infection; (5) there is no proof of the transfer of cancer from one human being to another by contact; (6) transplantation of cancer from one animal to another of the same species is well verified; (7) the production by any known parasite of epithelial proliferation analogous to cancer has not been demonstrated.

II. SPECIAL REASONS FOR BELIEF IN PARASITIC ORIGIN OF CANCER.

There occur in cancers, generally but not always within the cells, peculiar bodies, first described by Virchow, and considered by him to be evidence of endocellular cell division. These bodies are variously described, but the typical ones consist of a central dot surrounded by a relatively clear space, which in turn is surrounded by a membrane, often described as "double contoured." Of recent years these bodies have been closely studied, and, for various reasons, have been considered by some men to be parasites and the cause of cancer. These men believe that the bodies are parasites partly because of the supposed morphological resemblance to certain known parasites and partly because of what they believe to be an experimental production of cancers by inoculation of animals with the parasites. Some investigators believe that the bodies are blastomycetes or torulæ, others that they are protozoa.

1. *Blastomycetic Theory.*

The status of the theory that these bodies are blastomycetes and the cause of cancer can, in my opinion, be dismissed in a few words. The detailed reason for this opinion can be found in the previous report by the Cancer Committee of the Harvard Medical School. In that paper I drew the following conclusions from an examination of the results of the experience of other men and of myself. Examination of the results of the experience of other men and of my own experiments lead me to the conclusion that—

1. Certain blastomycetes can live and multiply in human and

animal tissues, produce local lesions and metastases in the internal organs, *i.e.*, they are pathogenic.

2. The lesions produced in animals by spontaneous infection with blastomycetes are acute inflammations, abscesses, or nodules of peculiar granulation tissue, and are not in the least analogous to cancers.

3. The lesions produced in human beings in cases of spontaneous infection with blastomycetes are acute inflammation (abscesses or ulcers), or proliferation of endothelium and connective tissue. At times a proliferation of the epidermis does occur, but is not due to the action of the blastomycetes, but is secondary to the chronic inflammation of the underlying corium. This proliferation of epidermis is not analogous to the proliferation of epithelium seen in cancers, since no epithelial metastases occur.

4. Blastomycosis in human tissues is very rare.

5. The lesions produced in animals by experimental inoculation with blastomycetes are, with the exception of Sanfelice's "successful" cases, inflammations or nodules of peculiar granulation tissue. Sanfelice's cases are not conclusive in themselves, are in direct opposition to the results obtained by all other observers, and, even if true, are logically explained as coincidences, and not as results.

6. Blastomycetes, as a rule, cause marked proliferation of tissue, and little infiltration with leucocytes, *i.e*, their toxic powers are small.

7. Blastomycetes primarily extend along lymphatic clefts and vessels.

8. Rarely in human beings, more often in spontaneously infected animals, and often in experimental animals blastomycetes may be taken into the blood vessels, disseminated throughout the body, and produce a general infection and metastases.

9. The secondary nodules have the same general character, *i.e.*, a formation of granulation tissue, as the original nodules.

10. The morphology of the so-called "cancer bodies" is not identical with that of the blastomycetes.

11. Blastomycetes are not constantly present in human malignant tumours and cancers.

12. Even if blastomycetes do occur in human cancers they are not present in such numbers and in such a relation to the anatomical lesion as to justify the belief that they are the cause of the disease.

All of these facts lead to the ultimate conclusion that there is no evidence that blastomycetes have anything to do with the production of human cancers.

The blastomycetic theory has rather lost its vogue of recent years, and in my opinion there has been no new evidence which would lead to any change of the above opinions regarding the relation of blastomycetes to cancer. On the other hand, since the appearance of my report, Foulerton and Price-Jones[1] have published a paper upon the bacterial examination of malignant tumours, which is in agreement with the results reported by Dr Oscar Richardson in the second report of the Harvard Cancer Committee, while Young[2] has reported observations on the pathogenic action of blastomycetes which confirm the correctness of my own observations. Foulerton and Price-Jones examined fifty-six malignant tumours, and concluded that "none of the so-called 'cancer yeasts' stood in any casual relation to cancer." Young inoculated animals with the blastomycetes of Sanfelice, Plimmer, and others, and concluded that pathogenic blastomycetes produced in animals' nodules composed of granulation tissue and blastomycetes, and that, although occasionally blastomycetes could be seen within cells which resembled cancer bodies, they were not the same as cancer bodies either morphologically or in their staining reaction. Moreover, Malvez[1] has shown that the serum of animals inoculated with yeasts has the power of agglutinating those yeasts, but Brauha[2] showed that the serum of cancer patients did not agglutinate the cancer yeasts, as they should have done if blastomycetes were present in, and the cause of, cancer.

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[1] Foulerton and Price-Jones: The bacterial examination of cases of carcinoma and sarcoma. "Rep. Cancer Research Lab., Middlesex Hospital," I., London, 1902.

[2] Young: The pathogenic action of blastomycetes. "Rep. Cancer Research Lab., Middlesex Hospital," I., London, 1902.

2. *Protozoon Theory of the Causation of Cancer.*

The evidence that protozoa are the causes of cancer can be divided into three heads. Some men believe that the cancer bodies are protozoa because of their alleged similarity to known protozoon parasites. Others have claimed to "cultivate" the alleged organisms, and to produce cancerous tumours by inoculating animals. Still others believe that a certain protozoon, the "plasmodiophora brassica," produces, in plants, cell changes analogous to the epithelial proliferation seen in cancers, and that therefore it is conceivable that the epithelial proliferation in cancer also may be due to a protozoon.

1. *Morphological evidence of parasitic nature of cancer bodies.*—Without going into a detailed review of the descriptions of the "parasite" by all of the different observers, a general statement of the conclusions drawn can be made. Thoma[3] described small unicellular organisms in the nucleus of epithelial cells of cancer, which from their size and shape and composition he believed undoubtedly were parasites, which might be the cause of cancer. Russell[4] described bodies, "fuchsin bodies," seen in the protoplasm of cancer cells, also in gummata, in fibro-adenomata, and in a "gelatinous sinus" of the knee. The bodies also were found in round cell infiltrations, in the connective-tissue stroma, and in the lymphatics. Russell described the bodies as spheres having a refractile capsule and staining intensely with fuchsin. He said that the bodies reproduced by budding or by the formation of spores. Ruffer and Walker[5] described bodies which occurred in the protoplasm of cancer cells, spherical, having a central nucleus, surrounded by protoplasm and a refractile capsule, often double. Similar bodies were seen by Sjöbring,[6] who said that they formed spores and cysts. Ruffer and Plimmer[7] described similar bodies, but saw no cysts. Similar bodies were described by Sawts-

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[1] Malvez: "Centralbl. f. Bakt.," XXIX., p. 688, 1901.

[2] Brauha: "Centralbl. f. Bakt.," XXX., p. 945.

[3] Thoma: Uber eigenartige parasitäre Organismen in den Epithelzellen der Carcinome. "Fortschr. der Med.," VII., p. 413, 1889.

[4] Russell: "Brit. Med. Jour.," 1890, II., p. 1356.

[5] Ruffer and Walker: Parasitic protozoa in cancerous tumours. "Jour. of Path. and Bact.," I., p. 198.

[6] Sjöbring: Ein parasitärer protozoaartiger Organismus in Carcinomen. "Fortschr. der Med.," VIII., p. 529, 1890.

[7] Ruffer and Plimmer: "Jour. Path. and Bact.," I., p. 395; II., p. 3, 1892.

chenko, Podwyssozki, Steinhaus, Foa, Roncali, von Leyden, Bosc, Schueller, and many others. Some men, Roncali and others, claimed to find such bodies in sarcomata. A study of the descriptions by the various men shows that one factor is constant in all of the descriptions, *i.e.*, the presence of small spherical bodies composed of a centre, a surrounding clear zone, and a refractile membrane, the latter often described as "double contoured." Beyond that point all agreement ceases. Some claimed that the bodies could be seen in the cell nucleus, but most agreed that the bodies came in the epithelial cell protoplasm. Some found them intercellular or in the connective-tissue stroma or in lymph spaces. Some men believed that the bodies reproduced by budding, others that they formed spore cysts and spores. Some claimed to find "sickle bodies." Various life cycles of the so-called parasite were worked out, no two agreeing on the successive steps. Besides these typical bodies a vast variety of other inclusions have been described. In fact any bizarre figure seen in cancer cells apparently was assumed to be one of the protean shapes which the parasite could take.

While many observers thus agreed upon the general appearance of the cancer bodies, the interpretation of their nature and their position in the animal world was very divergent. Some observers believed them to be blastomycetes. The status of the blastomycetic theory has been stated. Others believed that they were animal parasites and that they belonged in some one of the various classes of the protozoa. This divergence of opinion shows conclusively that their morphology is not sufficiently constant to allow any agreement as to their exact nature.

Other men, after a careful study of these cancer bodies, came, on a morphological basis alone, to the conclusion that they were not parasites. Some men claimed that the bodies were a result of cell degeneration, and Pianese's valuable monograph shows clearly that many of the endocellular bodies can be explained in that way. Other forms have been ascribed to the inclusion of red blood globules in the cells, or of polynuclear leucocytes. Borrel[1] believed that the typical bodies represented centrosomes. Dean[2] believed that the inclusions were masses of hyaline degeneration. Völcker[3] demonstrated that Schueller's "parasites" were cork cells accidentally present. It is noteworthy that in Labbé's Tierreich[4] all of these alleged parasites of different observers are included under the term "pseudo-coccidia." Greenough[5] advanced a theory which seems to me to explain better than any other the peculiar form and character of the typical cancer bodies. Greenough observed that the bodies occurred only in cancers arising in glandular organs and believes they are due to the secretory activity of the cancer cell. Tadao Honda,[6] after a careful study of the cancer bodies, agreed that they came only in glandular cancer; that they were not parasites, and that they represented the secretion of a hyaline substance.

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[1] Borrel: "Ann. de l'Inst. Pasteur," XV., No. 2, 1900, 49.

[2] Dean: Dr. Russell's characteristic organism of cancer. "Lancet," 1891, I., 768.

[3] Völcker: "Dtsch. med. Wochenschr.," 25th July, 1901.

[4] Labbé: "Tierreich." "Sporozoa." Berlin, 1899.

[5] Greenough: Cell inclusions in cancer. "Second Rep. Cancer Committee, H.M.S.," p. 128., "Jour. of Med. Research," VII.

[6] Tadao Honda: Zur parasitären Ätiologie des Karzinoms. "Virch. Arch.," CLXXIV., 1903.

So, on the whole, it may be said that from a morphological study of the cancer bodies no agreement has been reached as to their nature or origin, nor on a morphological basis is it likely that any proof of the parasitic nature of the bodies can ever be made.

2. *Experimental and "cultural" evidence of parasitic nature of cancer bodies.*—Schueller[1] claimed that by taking bits of perfectly fresh cancer, in which he found so-called parasites, and placing them in sterile test-tubes which were kept at body temperature, an increase in the number of parasites could be detected. This cannot, even allowing that his observations of the bodies were correct, in any sense be considered evidence of "cultivation," and, as has already been mentioned, it has already been shown that some at least of his alleged parasites were cork cells. Gaylord[2] inoculated a guinea-pig with ascitic fluid from a case of abdominal cancer, and believed that he produced an adenoma of the lung; he also inoculated a dog with ascitic fluid from another case of abdominal cancer, and believed that he produced disseminated nodules of cancer in the animal's liver. Allowing that the results were such as Gaylord claimed, the experiments offer no evidence of inoculation with a "parasite," for the possibility that cancer cells were present is not eliminated. Moreover, as has already been shown, competent men who have had ample opportunity to study his sections deny the accuracy of his primary observations.

3. *Evidence that known protozoa can produce in plants cell changes analogous to cancer in animals.*—In cauliflowers and other members of the cabbage family occurs a disease called by the Germans "Kohlhernie," in English "club-root," which is due to an infection of the plant cells by a protozoon, the plasmodiophora brassica. The organism appears as spores in the cells of the affected plant, and it has been claimed that the lesions produced in plants by these spores are analogous to the epithelial cell proliferation in cancer, and hence by analogy that the cancer bodies are protozoa and the cause of cancer. Animals have been inoculated with these spores to see if these known protozoa would produce epithelial proliferation. Podwyssotzki[3] inoculated rats and guinea-pigs, and produced "tumors" of mesodermal origin "similar to large-celled sarcomata or endotheliomata or the granulomata seen in leprosy," but did not produce epithelial proliferation. Some of the spores were seen in (phagocytic) cells. He thought that these endocellular spores looked like the inclusions seen in sarcomata (sic). Gaylord[4] inoculated animals with fresh club-root and produced nodules of granulation tissue, with but little infiltration with leucocytes after the twelfth day, containing many phagocytic cells in which were spores and spores cysts. No epithelial proliferation or cancer was produced. Gaylord[5] says that spores of the plasmodiophora brassica produce, in both cold and warm blooded animals, appearances "indistinguishable from that of cancer inclusions." This must apply to the appearance of the spore itself,

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[1] Schueller: Beitrag zur Ætiologie der Geschwulste. "Centralbl. f. Bakt.," XXVII., 14-15, p. 511, 1900.

[2] Gaylord: Loc. cit., supra.

[3] Podwyssotzki: "La Presse Méd.," 1900, 77.

[4] Gaylord: Plasmodiophora Brassica. "4th Annual Rep. Cancer Lab., N. Y. State Board of Health," 1902-3, 20.

[5] Gaylord: On the evidence of the nucleur division of certain cell inclusions. Loc. cit., 53.

since the spores are seen only in phagocytic connective tissue or endothelial cells, and that is no evidence whatever of the parasitic nature of cancer bodies, for exactly the same statement as regards morphology can be made in regard to occasional faintly staining endocellular blastomycetes. Von Tubœf[1] has shown that the so-called "cancers" of plants may be caused by animal or vegetable parasites or by frost, and cannot be compared with human cancers. When due to a plant fungus the parasite is well known and of large size, and unlike the cancer bodies. He inoculated animals with plasmodiophora brassica and produced nothing analogous to cancer.

On the whole as regards the protozoon theory, it may be said that a settlement of the question on a morphological basis alone is very unlikely, especially in dealing with a disease the incubation of which is unknown, and the early stages of which so seldom can be studied. For, in dealing with microscopic spheres of not too constant form, it will always be possible to confuse them with, or see resemblances to, various simple lower forms of both plant and animal life which also have a spherical shape. Unless a definite and constant cycle of stages can be seen which closely resemble the life cycle of known vegetable or animal parasites, conclusions based upon morphology alone must always be doubtful, especially in a disease like cancer, the infectiousness of which is not proved. Lubarsch has said that for those who claim that the cancer bodies are parasites, it is necessary to prove not merely that the bodies resemble known parasites, but that they cannot be anything else. This statement is emphasized by the fact that certain observers have considered the cancer bodies at one time to be protozoa and at another blastomycetes, and vice versa. The constancy of form of the bodies is not such as to allow as yet any agreement upon their alleged life cycle.

Moreover, there is no proof that any known protozoon produces a proliferation of epithelial cells analogous to the epithelial proliferation in cancer. Nor has there been any verified, indisputable, or convincing proof of the production of cancer by any of the so-called protozoon cancer parasites. Nor has there been any successful isolation, cultivation, and inoculation of any cancer protozoon in accordance with Koch's law. As regards protozoon parasites it must be acknowledged that as yet it is practically impossible to present proof of their parasitic nature in the way that is demanded in the case of bacterial parasites, and certain diseases are accepted as due to a protozoon parasite without complete compliance with Koch's law, but in the case of the so-called cancer parasite the alleged cultural and inoculation experiments are so imperfect as to exclude even the probability of their being the cause of the lesions they are alleged to have produced. The same reasoning applies with redoubled force to the blastomycetic theory because it is a simple matter to cultivate blastomycetes outside of the body, and yet in no case of alleged cancer production has Koch's law been complied with.

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[1] Von Tubœf: Ueber Krebs bei Pflanzen. Verhandl. des Komités für Krebsforschung. Heft, i, 74, Berlin. 1902.

(*To be continued.*)

ABNORMAL GENITAL ORGANS IN THE DOMESTICATED ANIMALS.

By JAMES F. CRAIG, M.A., M.R.C.V.S., Professor of Anatomy,
Royal Veterinary College of Ireland, Dublin.

AMONG the lower animals malformations of the genital organs are sometimes observed, and, indeed, cannot be regarded as being very rare. The form which the abnormality of these organs takes is very various, but perhaps the most frequent of all is hermaphrodite. The cause or causes of these variations from the normal cannot be said to have been in any way clearly demonstrated, but that they do occur is not at all surprising. When we consider the complex processes and stages through which the embryo passes on its way from ovum to fœtus, the wonder is not that there are so many abnormalities found, but that they do not more frequently occur. It is not as if even from the beginning a male and female were quite different in form and appearance. In the ovum no one could distinguish between the two sexes, and it is not until a considerable period has elapsed after the process of development has begun that the sexes become to some extent differentiated. Even when the adult stages are reached there are organs connected with the genital apparatus present in one animal which are homologous with organs possessed by the opposite sex. Thus, in the adult male there is to be found a small bladder-like body placed on the dorsum and towards the neck of the urinary bladder, and opening into the roof of the urethra, called the uterus masculinus, which is the homologue of the female uterus. In the adult female a small erectile body, called the clitoris, is present within the lower commissure of the vulvar opening, and this is looked upon as the representative in the female of the male penis.

These organs, appendices of the genital apparatus, which the male has in common with the female, are normally very small—in some cases so small that they are very difficult to find, even when making a careful dissection. Occasionally these bodies assume very large proportions, and take on many of the characters of homologous organs in the female.

The following examples of such abnormalities have been brought before my notice through the kindness of Principal Mettam, of the Royal Veterinary College of Ireland:—

Case I.—One set of genital organs which I examined was taken from the carcase of a bullock slaughtered at the Dublin abattoirs. This animal was described as possessing an external genital apparatus which would have led one to suppose that it belonged to the male sex. The testicles, however, were not present in the scrotum. When the abdominal cavity was opened these organs were found attached to the roof of the abdominal cavity, at the entrance to the pelvis, each by means of a short double fold of peritoneum, and in the position of ovaries. The testicles themselves are abnormally small, irregular in shape, flabby in consistence, and of a light pink colour. The right testicle, somewhat pear-shaped, is larger than the left, and attains the size of a walnut. The left testicle reaches the size of a hazel-nut.

Above and towards the anterior extremity of the testicles, and almost concealing them, are to be seen the large and tortuous spermatic vessels, well marked off from the surrounding fat by their deep red colour. From the anterior extremity of the testicle there springs a small tube, which courses backwards along its upper border, coiled up and arranged in the form of a column, somewhat after the nature of a small epididymis. This tube is continued backwards from the testicle in the double fold of peritoneum by a very fine flexuous vessel, the vas deferens, and this latter, difficult at any time to trace within the peritoneal band, becomes lost, after a course of a few

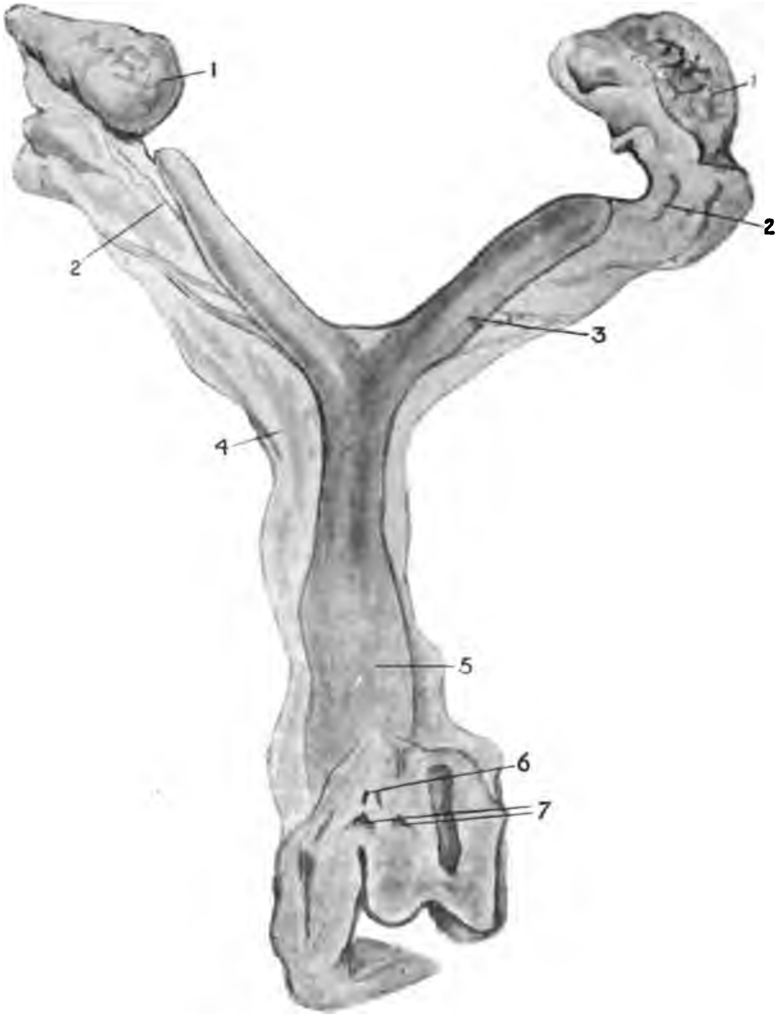


FIG. 1.

Genital Organs of Bovine Hermaphrodite (Case I.).

1. Testicle; 2. Vas deferens; 3. Right cornu of uterus masculinus; 4. Double fold of peritoneum; 5. Body of uterus masculinus; 6. Urethral opening of uterus masculinus; 7. Urethral opening of the ejaculatory ducts.

inches, in a mass of fat. The peritoneal folds above mentioned fuse with one another in their posterior half, and are noticed to contain not only these testicles and vasa deferentia, but also a body which has assumed the character of a female uterus. This organ consists of an irregularly cylindrical tube, about 1½ inches in diameter and 8 inches in length, broadest near its middle, placed in the middle line between the two vasa deferentia, bifurcating anteriorly, and opening at its posterior extremity by a minute aperture on the summit of an eminence on the roof of the urethra, called in the male the veru montanum. Each anterior bifurcation follows the course of the vas deferens of one side, and has the form of a cylindrical tube, about half an inch in diameter and 4 inches in length, blind at its anterior extremity, and posteriorly opening freely into the lumen of the main tube. This organ, with its anterior portions, resembles in appearance the letter **Y**. It is filled with a thick, transparent, mucous fluid. The walls of the organs are thick, and the inner lining is smooth and rose-pink in colour. On either side of the opening of this body into the urethra a large aperture, in which even an ordinary lead pencil could be inserted, is to be found. These openings lead into very short blind tubes, and they represent the urethral outlets of the ejaculatory ducts.

The accompanying illustration (Fig. 1) shows the appearance of the genital apparatus above described.

In this instance the genital organs possessed by the animal were essentially male. The animal was a cryptorchid, and, as is common in these cases, the testicles were small and flabby. The uterus masculinus, however, had attained the proportions of a well-developed female uterus.

Case II.—A few days after the organs above described were found, a similar case was observed in an animal of the same species which had been slaughtered at the same abattoirs. In this case the organs were attached in the abdomen and pelvis, as in the previous instance. The testicles are also abnormally small, the right one again being the larger. The vasa deferentia are very much thicker and better defined than in the first case, reaching the thickness of a goose quill. Anteriorly there are well marked globus major, body, and globus minor of the epididymis. Posteriorly each vas deferens becomes very much dilated into a bulbous portion, but unfortunately, owing to the fact that the specimen I received had been cut off short, the vas deferens cannot be traced to its posterior ending. Still attached to the specimen are the two glandular vesicular seminales of the ox, at each side of the posterior single tube of the uterus masculinus. This latter organ presents a very similar appearance to that of the one previously described.

Fig. 2 gives a very good idea of the appearances presented.

Case III.—Another specimen showing abnormal genital organs was removed from the carcase of a pig, and forwarded to the College by Mr J. Maguire, M.R.C.V.S., Lisnaskea. In this case the animal was said to present externally all the normal appearances of a sow, possessing normal mammary glands and external vulval opening. Indeed, it resembled a sow so much that it had been sent to the boar three times, but as it would not breed it was fattened and afterwards slaughtered.

When the genital organs were removed from the animal after death, it was found that the uterus was as well developed as one would look for in an aged sow, consisting of two long, convoluted, cylindrical horns, fused together at their posterior extremities to form the short body of the uterus, the latter being continued to the vulval opening by a narrow vaginal tube. In the position of the ovaries were placed organs which in no way could be distinguished from testicles by a naked-eye examination. These are ovoid in shape, about the size of duck eggs, pinkish in colour, having a smooth exterior, the course of the blood-vessels being observed through the

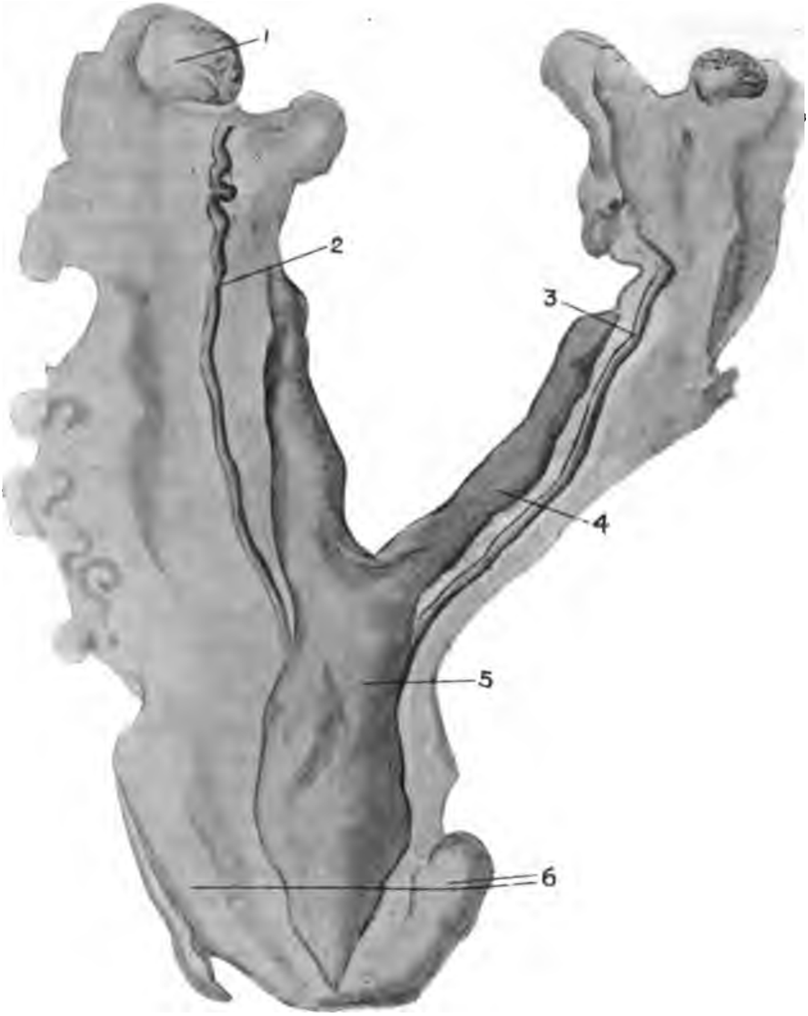


FIG. 2.

Genital Organs of Bovine Hermaphrodite (Case II.).

1. Testicle; 2 and 3. Vasa deferentia; 4. Right cornu of uterus masculinus; 5. Body of the same organ; 6. Vesiculæ seminales.

semi-transparent covering of the organ. At the anterior extremity of the testicle a very much convoluted tube begins, and arranges itself along the dorsal border in the form of an epididymis. This tube is then continued by a slightly flexuous vessel, which is only an inch or two in length and about the thickness of a goose quill. This vessel opens into the anterior extremity of one cornu of the uterus, much in the same way as the normal Fallopian tube of the sow. The line of demarcation between the tube and horn of the uterus is not well marked (*see* Fig. 3).

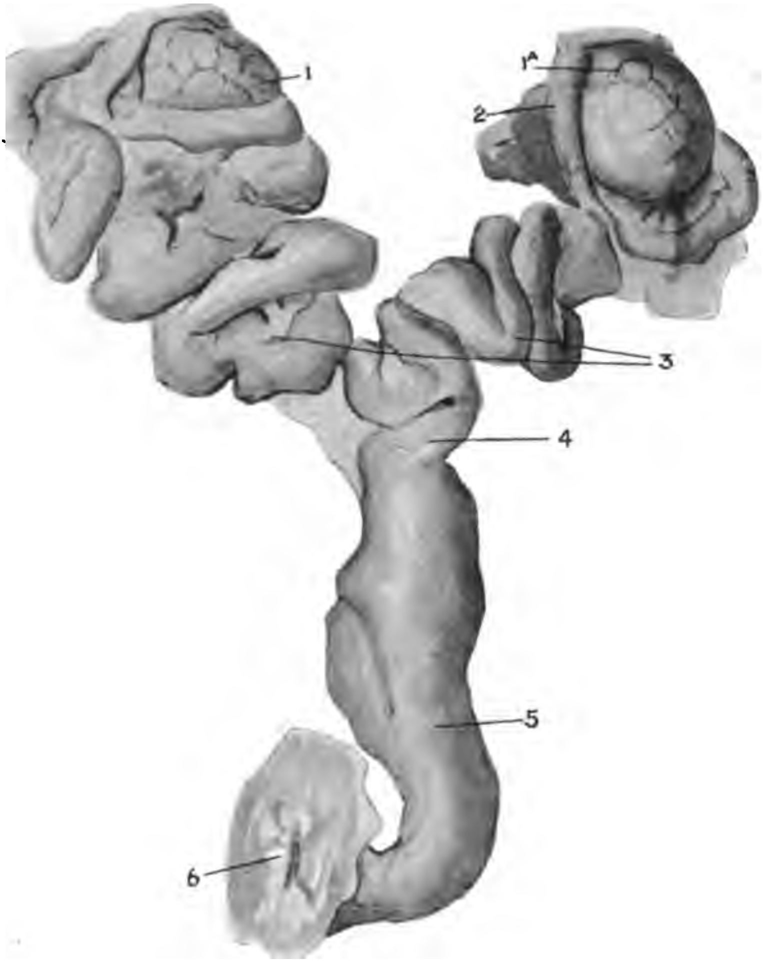


FIG. 3.

Genital Organs of Hermaphrodite Pig (Case III.).

1 and 1A. Testicles; 2. Epididymis; 3. Uterine cornua; 4. Body of the uterus; 5. Vagina; 6. Vulval opening.

Microscopic Examination of the Testicles.—Although the essential organs of the genital apparatus in Case III. have been referred to as testicles, and presented the ordinary appearances of these bodies, the histology is not that of normal testicles. I removed small pieces of these organs and prepared microscopic sections of them. The accompanying photograph (Fig. 4) shows the nature of the tissues under observation. The histology of the organ is certainly remarkable. On the outer side of this body there is placed a layer of serous membrane, and under this a well-marked fibrous covering. Within this tunic the main substance of the organ is situated. The groundwork of the tissue is extensive, and is essentially cellular in character, the cells being large, polygonal in shape, with well-marked oval nuclei and more deeply stained nucleoli. These cells appear very much like the cells found in the corpus luteum of the ovary. In the sections this matrix appears to be divided into lobules, the divisions being formed by a network of open spaces. Probably these open spaces were produced in the process of hardening the tissue with consequent shrinking of the cell protoplasm, so that the apparent lobulation is really artificial. Scattered throughout the matrix and cut in the sections in different ways are large numbers of tubules, bounded on their outer side by a basement membrane consisting of a single layer of flat cells. Within this basement membrane the chief cellular layer of the tubules is placed, and is composed of a single stratum of short columnar cells, of which the nuclei are large and oval and stain deeply. The lumen of each tube contains some homogeneous material, staining in the same way as the cell protoplasm, and arranged in the form of a network. The spaces in

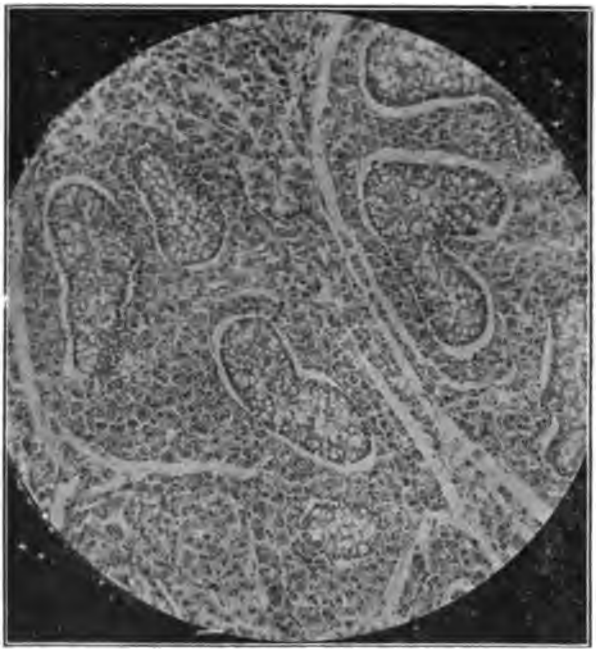


FIG. 4.

Section (×105) from Testicle of Hermaphrodite Pig (Case III.).

the network are rounded, and look like vacuoles. No doubt they were formerly occupied by fat droplets. The material produced by the cells of the tubule had evidently undergone fatty degeneration.

What, then, is the explanation of this abnormal appearance? This is certainly not the histology of the testicle, far less that of the ovary. The tubules seen in the specimen no doubt represent the degenerating seminiferous tubules of the male testicle. The cellular groundwork can be understood as an enormous development of the Wolffian body, or of those cells or groups of cells normally to be found in the testicle at places between the tubules.

In Case III. the greater portion of the genital apparatus, then, is female, but the essential organs, on naked-eye inspection, might be regarded as male, although the histology does not show them to be normal testicles. How can we understand the appearance of this condition? I have already said that for a certain time after the process of development from the ovum has begun the male cannot be distinguished from the female. This is the so-called indifferent stage. At this stage there are formed two ridges, one on either side of the roof of the abdominal cavity. Each ridge consists chiefly of a Wolffian body and Müllerian duct. The Wolffian body itself is formed of a number of Wolffian tubules and a Wolffian duct which lies alongside the Müllerian duct. This apparatus evidently acts as an excretory organ to the embryo. It is from these bodies that the genital organs are formed. Now, in the development of the male

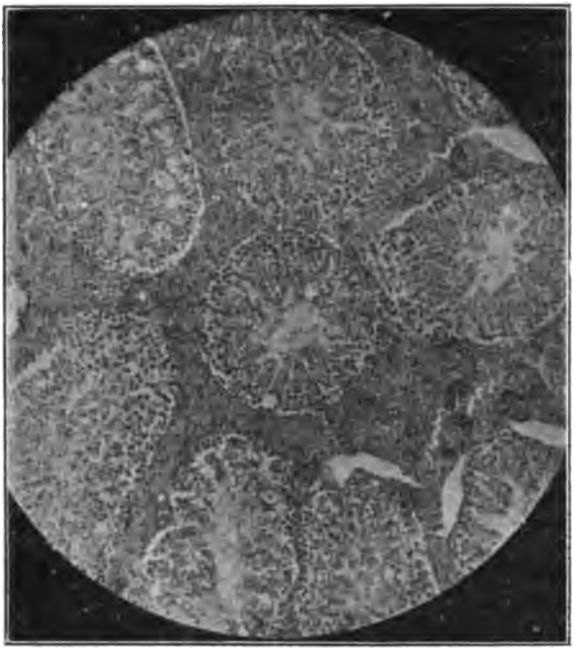


FIG. 5.

Section (×105) from Normal Testicle of Boar, for comparison with the preceding figure.

the anterior portion of the genital ridge gives rise to the testicle, and the Wolffian duct forms the vasa efferentia, epididymis, and vas deferens. The Müllerian duct aborts, and of it in the adult male the chief part that remains is the uterus masculinus. In the development of the female, on the other hand, the ovary is formed from the anterior portion of the genital ridge, and the Wolffian duct atrophies, all that is left of it in the adult female being found at the fimbriated opening of the Fallopian tube. The Müllerian duct becomes the genital duct of the female, forming the Fallopian tubes, the uterus, and vagina.

What has occurred in this instance (Case III.) is that the anterior part of the genital ridge has become developed into a testicle, the Wolffian duct has persisted and has formed the vasa efferentia and epididymis, and the Müllerian duct has given rise to the appearance of the Fallopian tube and well-developed uterus. The Fallopian tube has then become continuous with the epididymis.

With regard to the peculiar appearance of the testicle, the degenerate condition of the seminiferous tubules has probably been the result either of inaction or an imperforate condition of the ducts of the organ. The so-called "interstitial" cells, which are comparatively very abundant in the testicle of the boar (*see* Fig. 5), have received a stimulus to propagate, and have taken advantage of the atrophy of the seminiferous tubules. This has then given rise to the remarkable abundance of the cellular groundwork in the substance of the testicles.

SOME POINTS TO BE CONSIDERED IN CONNECTION WITH RHODESIAN REDWATER.

By STEWART STOCKMAN, Late Principal Veterinary Surgeon to the Transvaal Government.

IMMUNITY.

THAT natural immunity from Rhodesian Redwater can be acquired by a race of cattle seems beyond dispute, as witness the power of resistance to the disease possessed by cattle indigenous to German East Africa. This resistance, however, has in all probability, taken a century or more to acquire. Concerning its exact degree and quality we are still without exact information. For example, we do not know whether cattle of the East Coast are born absolutely immune, or whether they simply take the disease in a mild form, recover, and thereafter completely resist, as we see in the few animals which have become salted in the Transvaal. This information acquires the highest importance in view of the possibility of it being found advisable to restock partially infected areas with cattle from the East Coast. One has already seen the advantage of restocking the Transvaal with cattle from Texas and Queensland. These cattle are born in infected areas and have acquired a certain degree of resistance to Texas Fever—ordinary Redwater—which exists practically all over the Transvaal, and the death rate amongst them has been very materially less than that experienced with other imported breeds. The case of the two diseases, however, is by no means parallel. Texas Fever is permanently established in the country, whereas we devoutly hope that Rhodesian Redwater is not. The former disease is much less fatal than the latter, and a large proportion of the progeny of imported animals acquire great resistance towards it, by picking up the infection at an early age. Our experience with Rhodesian Redwater, however, so far shows that at least the first generation of calves, even those born from salted cows, die of the disease if brought in contact with infection. It has not yet been determined whether a calf born of a mother which suffered from a non-fatal attack of the disease during her pregnancy is highly resistant for a certain time after birth, as happens in the case of certain bacterial diseases, but the question loses much of its practical importance in a newly invaded country, where over 95 per cent. of the affected animals succumb, and the unborn calves never see the light of day. Another very important difference between the two diseases is, that while animals salted against ordinary Redwater permanently keep up the infection in a country, all the evidence goes to show that those salted against the other disease do not do so. This, of course, is a point in favour of Rhodesian Redwater, but in a newly invaded country it is exceedingly inadvisable to treat so-called salted animals differently from others, as the evidence upon which the majority are said to be salted is quite unreliable, and in most cases incapable of proof, unless they be experimentally exposed with control animals on ground known to be highly infected. Certain stock owners assert that they possess salted animals, but upon inquiring into the matter one often finds that the number in their possession is out of all proportion to established facts concerning the death rate. As the question is rather important, it might be as well to examine the evidence upon which this assertion is usually based.

Cattle which have recovered from the disease certainly acquire immunity, but in the case of many so-called salted animals the owners cannot definitely say that their animals ever suffered from Rhodesian Redwater, while in others it is clearly stated that they did not. It is simply concluded that the animals are immune because they are alive. We know, however, that cattle may run on infected pastures for months without picking up infected ticks, and we have several times observed this in the case of a few survivors from a herd which has been decimated. Still, it does not necessarily follow that the survivors are immune. It often simply means that the fewer the animals wandering over an infected farm—of 6000 acres in some cases—the less likely are the infected patches to be encountered; we know of cases where a solitary survivor has lived for months, and finally succumbed to the disease. On the other hand, we have seen a single animal of a batch, all of which we were absolutely sure had never been exposed to infection until we did so purposely, live practically right through a tick season on our experimental ground at Nelspruit, where its companions died in the usual time. This animal's temperature was regularly taken every day during exposure, and smears made from its blood were repeatedly submitted to microscopical examination. Therefore, we can be certain that it did not at any time suffer from the disease. It may be, then, that there exist even outside the home of the disease a few individual animals which for some unknown reason possess a high degree of resistance to it. Indeed, it is very probable that this is so, for we know it to be true of nearly every other disease ; that is to say, one will always observe, both in human and animal plagues, that there are individuals who resist in spite of the fact that they have been constantly exposed to infection. Whether this is a provision of nature to prevent a race being totally wiped out by plagues, it is difficult to say, but we do know that the percentage of individuals endowed with this natural resistance is so small that we dare not allow it to enter into our calculations regarding such economic problems as the re-stocking of infected areas, more especially when we are aware that one can only select the individuals by placing very large numbers in actual contact with infection. One of the methods adopted by farmers for localising salted animals, with a view to purchase, is to proceed to a native location where the disease exists, and observe the number of hides. If the hides are numerous, they conclude that the place is badly infected, and, therefore, that all cattle still alive must be salted. One cannot but admire the ingenuity of this deduction, but, unfortunately, it is drawn on an insufficiently intimate knowledge of the disease. It has already been explained that mere survival is not an adequate guarantee that the animals are salted, and enquiry has several times brought to light that the number of reputedly recovered animals, when compared with the numerical strength of the herd before it became affected, was far in excess of the percentage which may be expected to survive an attack of the disease. Further, it should be understood that natives, of all persons, have adopted the principle of changing the pastures as soon as they have reason to believe that contagion exists in their herds, so it is quite possible that the large proportion of survivors from a disease like Rhodesian redwater is to be explained in this way.

We come back to the question of whether cattle of the East Coast are born absolutely immune, or whether they are simply endowed with a high degree of resistance which becomes converted into absolute immunity by an attack of the disease, from which they suffer but slightly. This question can only be investigated in the home of the disease, and it should not be a very difficult one to solve. Reasoning from analogy, the second explanation seems the more probable. If, for example, we study the question of rinderpest in the plains of India, where it has existed for centuries, we find that the cattle are highly resistant, though not absolutely immune. So slightly do many of them suffer from the disease that an attack might easily escape the observation of an expert, unless he had the animals under the closest observation. The writer experimentally injected virulent rinderpest blood to eighty animals of the plains of India with only one fatal result, while many of the animals so injected showed nothing more than a passing fever. Nevertheless, we know that if these animals are brought into contact with others which have not acquired this natural resistance, cattle of the hills for example, or those from the western hemisphere, they fatally infect the latter with rinderpest. The continual existence of these mild and imperceptible cases renders it well nigh impossible to eradicate the disease. It must not be forgotten, however, that rinderpest is a disease which is directly contagious, and possibly, therefore, not exactly comparable with Rhodesian redwater. Still, the broad question of natural immunity is a general one, and may be discussed as such. Moreover, we know that cattle acquire a high degree of resistance to the indirectly contagious disease known as Texas fever —ordinary redwater—when they are reared in an infected country. This resistance to Texas fever, however, is hardly an immunity in the proper sense; it would be better described as a tolerance to the causal parasites, which persist indefinitely in the blood. An attack of Texas fever does not result in the animal organism becoming an absolutely unsuitable medium for the growth of the parasite. On the contrary, if it remains in an infected area, it becomes repeatedly reinoculated by ticks without suffering much harm; blue ticks can infect themselves by sucking its blood, and the blood if injected directly into a susceptible animal gives the disease to the latter. In the case of Rhodesian redwater, however, it has already been mentioned that all our experimental evidence goes to show that ticks cannot be infected on recovered animals. It would seem, then, that in the latter disease an attack brings about a condition which prevents the infecting agent gaining a second footing. In this important respect the immunity acquired against Rhodesian redwater seems to meet that developed towards rinderpest. It points also to the possibility of each being due to the same chain of circumstances, viz., a high degree of inherited resistance, which can be converted into an absolute immunity by a mild attack of the disease. As before indicated, the importance of settling this question is paramount, in case it should be deemed advisable to restock partially infected districts from the East Coast, and it should not be very difficult to solve by investigations conducted on the spot.

If, for example, it should turn out that the individuals of the truly indigenous race are born with absolute immunity, restocking with that breed ought not to be attended with much danger. It would hardly be possible, under the circumstances, for such animals to be the means of importing infected ticks—the latter cleanse themselves on non-susceptible animals—but the last element of risk would be removed by dipping them on landing, and keeping them in kraals for sufficient time to allow all ticks to drop off.

If, on the other hand, the indigenous race is only partially resistant, one would run considerable risk of re-importing the disease, and the importation could only be done safely by subjecting the animals to a lengthy period—six weeks—of quarantine in kraals. This, of course, would be an expensive undertaking for the importer, as the cattle could not be allowed to graze during the period. Moreover, although the quarantine period would remove the risk of importing the disease, it would not, of course, affect the question of whether animals are absolutely immune or only partially resistant. In the latter case, the calves born from the cows and a certain percentage of the imports themselves would be liable to suffer from the disease on coming in contact with infected ground, and although the death rate might possibly be inconsiderable, the result would be permanent infection of the pastures, and the impossibility of importing cattle from elsewhere, or exporting to other districts which are clean.

I think it necessary here to point out that one might be inclined to consider that the question just discussed was settled by the fact that the disease was brought to Portuguese territory and to the Transvaal by cattle imported from German East Africa. We have reason to believe, however, that the imported cattle were not all of the race indigenous to the infected zone, but were composed chiefly of other breeds, which had been trekked through it for shipment at Dares Salaam.

On account of the economic importance of the question just discussed, I think steps should immediately be taken to solve it, even if it means sending an expert commission to the East Coast to investigate the matter.

POINTS TO BE CONSIDERED IN SEEKING THE ORIGIN OF OUTBREAKS.

The assistance which one may derive from the permit system in seeking the origin of an outbreak is undeniable. Most of the farms, however, were infected before the system was introduced, and not a few have since been infected, although no permits have been issued for movements on or off the place. In the former cases, one got reports sooner or later that the cattle were dying of a somewhat mysterious disease, but only in a few cases could reliable information be obtained regarding recent movements of cattle and the origin of the initial outbreak; one could only make enquiries all round the neighbourhood, with the object of finding out whether any suspicious deaths had occurred of late and watch for others.

Where cases of sickness were discovered, blood smears were taken from the animals and examined for piroplasmata. In this way a good many old outbreaks were located, and during the last eighteen months we have acquired a pretty complete knowledge regarding infected routes and farms. In the latter cases, *i.e.*, where farms have been infected since the introduction of regulations, the matter of tracing the origin of outbreaks became somewhat easier, as many of the people were by that time more alert, more suspicious of moving cattle, and readier to report, while continuous investigations supplied us with further details regarding the mode of spread. It is still, however, often exceedingly difficult to arrive at the exact truth, and one must rely, to some extent, on deductions.

In conducting an enquiry into how a farm became infected, in the absence of positive information from the owner, one must consider the following points:—

1. That a farm can only have been infected by a sick animal or animals having been brought on to the place.

2. That the sick animal if it remained on the place must almost certainly have died thereon, as the disease is very fatal, and that a resident is pretty certain to know whether a death has occurred or not.

3. That infected ticks may have been dropped by a sick animal whilst merely passing over the place, but in this case the infection of the ground is not likely to be great; the deaths in the first outbreak will, therefore, be few, whilst it will sometimes be possible to trace the infecting animal by hearing of its death elsewhere and obtaining from the owner details of the route over which he passed.

4. That if the sick oxen have been on the farm for about fifteen days (see incubative period) before becoming sick, it is practically certain that they picked up the disease on the spot.

5. That while it is theoretically possible for the disease to jump about 200 miles by road, experience has taught us that it seldom travels more than 70 miles at a jump, and generally less (see former remarks on this subject).

In the absence of exact knowledge on the part of the owner, regarding where his cattle which have recently been travelling, picked up the disease, one can sometimes trace the probable source of infection by working on this line; it sometimes results in the discovery of an unreported outbreak. Moreover, sick animals which have merely passed over a farm and infected it may occasionally be traced in the same way.

6. That in the case of animals which have become affected whilst travelling along the road, if one can obtain the approximate date upon which they became sick and the particulars of the various treks, it will be possible to deduce from the incubative period of the disease —average ten days—the probable region in which infection was picked up, and one is enabled to pursue one's search for infected centres, in a more or less restricted quarter, if they are not already known. For example: let us suppose that oxen have become sick five days after their arrival at a certain place. The incubative period being about ten days, one may conclude that infection was picked up somewhere in the quarter where they were about ten days before arrival. Similarly, if one hears of a suspicious death on a farm too late—as often happens in South Africa—to make a satisfactory *post-mortem* examination of the case, one should search around the quarter in which the animals were located twenty days or so before; that is to say, in the case of a death, one must reckon with the incubative period—about ten days—plus the duration of the disease—about ten days—in order to arrive at an approximate idea of where the disease was picked up.

7. That if sick animals have passed along a road it is likely to be infected; it should be watched and, if possible, closed to oxen.

8. That in the case of first outbreaks on new ground, the number of infected animals is generally few, because under ordinary circumstances only a comparatively limited number of infected ticks are brought on to a farm in the first instance. At each successive outbreak, of course, a greater number of ticks are infected, and the deaths become more numerous. If, then, disease is reported on a farm, and one finds a considerable number of sick animals, the following conclusions suggest themselves: That the said farm was infected for some time before the report was made; or that the cattle have recently been moved from badly infected ground; or that the animals during a recent trek have outspanned successively on infected ground. Reasoning from the law of chances, however, and the geographical distribution of the disease two years after the date of invasion, the latter suggestion is the most unlikely of the three. I know of only one road—Pretoria to Rustenburg—along which the majority of the farms bordering upon it are infected for a distance of sixty-five miles, and, in order to justify the third conclusion, one would be compelled to assume that each time the animals were outspanned, they had the

bad luck to graze over the actual patches of infection on successive farms. This, of course, is not possible.

9. That when a pasture is infected for the first time by one or two sick animals being brought on to it or having died upon it, one cannot expect a second outbreak from ticks infected on the first victims until a month or six weeks after the first case of sickness. The delay, however, may be much longer. The reasons for this are easy to understand if one considers the life history of the brown tick, the manner in which it becomes virulent, and how animals pick up infection. It will be remembered that the intermediate stages of ticks take up the causal agent—piroplasma—of the disease from the blood of sick animals; and it is not until they have moulted and passed into the next stage that they are able to infect other cattle. During the period occupied in moulting it seems highly probable that the piroplasma itself undergoes a development inside the tick, for one has to explain the fact that the blood of a sick ox does not do any harm when injected into another, although it contains parasites which fatally affected the former. Possibly the piroplasmata in the blood are asexual forms of the parasites, reproducing themselves by fission only. It is probable that inside the tick they become sexually mature, that the sexes unite, and produce a living cell which in the body of the ox gives rise to other cells capable of multiplying asexually by fission. In the case of ground infected through virulent ticks having been dropped off a sick animal, however, one cannot always count on the ticks causing an outbreak about six weeks afterwards. If the season is unfavourable it may be several months before the moulting is completed, and even then the oxen on the farm may miss the infected patches for months, or may never come in contact with them at all.

For the above reasons, one must not come hastily to conclusions regarding outbreaks which are based on the moulting period of the ticks. Similarly, one cannot assert that a quarantined farm is free from infection simply because no case of disease has occurred upon it for several months.

10. That although the movement of sick cattle is for all practical purposes the only important factor in the spread of this disease, there are other possibilities which might come into play. Grass, hay, or bush, cropped off an infected area may contain virulent ticks, and might be the source of infection at a remote place. Regulation 44, Section (*d*) of Government Notice 834 of 1903, forbids the removal of grass or hay from an infected place, except by permit. We have never in the Transvaal, however, obtained evidence to show that any outbreak has originated from the transportation of grass; indeed, we have generally been able to trace infection to the movement of sick cattle, or we have found very strong presumptive evidence of the same. Still, in discussing this matter with Mr Gray, C.V.S., Rhodesia, he informed me that he has met with one or two cases of the disease in stabled cows, and he was forced to assume that the ticks had been brought into the stable on grass. This factor must be looked upon as a distant possibility, which might acquire greater practical importance were it more customary to transport grass or hay from one farm to another. There is also a chance of the disease having been carried by the hides of its victims, and for this reason, Regulation 44, Section (*e*)

of Government Notice 834, forbids the removal of the hides except after disinfection. The ticks however, soon leave the body of a dead animal. Moreover, the fact of a tick being attached to an animal at the time of its death generally means that it has not completed its engorgement, and, should it be carried away on a hide, the chances are very much in favour of the interruption in its life cycle proving fatal to its further development. In following up the origin of an outbreak, then, one must not attach too much importance to the possibility of the disease having been spread by the transportation of green hides. Two authentic cases have come to my notice where an abortive attempt was made to maliciously infect a neighbour's ground by the aid of green hide. In each case a tail was cut off at the root, and deposited in the kraals. An attempt of this kind, might, of course, be successful if the tail were removed immediately after death, because it might just so happen that one or two brown larvæ or nymphæ on the point of complete engorgement were attached to the tail at the time. The chance, however, is not particularly great, and I very much doubt if the perpetrators of these attempts were really aware of the ingenuity of their device. I am inclined to believe, rather, that they were guided simply by the knowledge that ticks carried the disease, and the sight of the common though harmless red ticks at the root of the tail. To pluck incompletely engorged brown ticks off a sick beast and disseminate them on another pasture is not at all likely to result in infection, as the ticks thus treated, seldom, if ever, bite on another animal. Still, it would certainly be possible to disseminate the disease by scattering infected ticks on clean pastures. To do this, however, a decree of expert knowledge which only a few possess would be necessary. As malice is a factor in the sanitary police of animal diseases which, unfortunately, one must, at least reckon with, it is inadvisable to put this knowledge at the disposal of all and sundry by further discussing the subject in detail.

11. That animals other than those of the bovine species could only carry infection by the merest chance.

Once an infected tick has bitten an animal, it apparently discharges its poison, since it afterwards fails to infect others. Should it in the first instance bite on a healthy ox, the latter, of course, gets the disease and serves to infect other ticks, but the poison of the infecting tick is discharged. If, however, the tick bites on, let us say, a mule, the poison is again discharged, but the mule does not get the disease, and therefore cannot infect other ticks. Still, we know that ticks often do not bite on their host for some little time after they have crawled on to its body. One can imagine, then, the remote possibility of one of these infected ticks being transported a short distance on a mule and getting knocked off before it has bitten. One can also imagine that infected ticks might be transported on the clothes of human beings.

In the transvaal and Rhodesia, however, no restrictions whatever have been put on the movements of animals other than bovines, and no outbreak has ever been traced to men, sheep, goats, horses, mules, or donkeys during a period of about three years, although they have been freely moving from badly infected areas.

12. That all the evidence yet available goes to show that the disease is not carried by animals which have recovered from the disease, as in the case of ordinary redwater. One must, however, always remember

that very few recovered animals exist, and one must accept with the gravest suspicion all statements concerning animals being salted, especially if the number is out of proportion to the accepted percentage of recoveries; under five per cent. recover, according to our present statistics. We have never been able to experimentally infect ticks by placing them on salted animals, although it is easy to do so by allowing them to suck on sick oxen. Moreover, salted cattle have been allowed to graze for months on a clean farm along with green cattle, and yet no outbreak has occurred.

The above correlation of observations will be found useful in tracing the source of outbreaks. Especially will this be the case in a country where movement of cattle is allowed only by permits of which records are kept.

In the still clean colonies of South Africa threatened by invasion, and in those only slightly infected, many of the conditions which provided the material for the above synthetical and analytical examination of affairs as they stand need never arise, as we to-day possess a reasonably full knowledge concerning the pathology of the disease. It must be remembered, however, that this new disease had obtained a firm footing in the Transvaal, before its pathology was properly known. Moreover, before it was found possible to organise any preventive measures, it was already advancing in an interrupted line extending south-eastwards from near Pietersburg in the north, through Middelburg, Lydenburg, and Barberton Districts, to a point slightly west of the Swaziland border, and from there southwards almost to the Natal boundary. Under these circumstances a working theory had to be gradually constructed upon which to base our investigations into the origin of outbreaks when exact and reliable information was not forthcoming from the owner of the animals. I think it is possible to prevent the disease spreading to the Colonies neighbouring on the Transvaal, and if I may presume to offer advice to the former it would be that, in addition to all other precautions taken, one cannot well afford to neglect the establishment of a buffer zone into which ox transport is forbidden, and inside which movements of cattle are only allowed by permits of which records are kept.

EDITORIAL ARTICLE.

THE IMPROPRIETY OF PASSING THE CARCASES OF ANIMALS SLAUGHTERED WHEN MORIBUND.

THE various Public Health Acts in force in this country contain provisions which relate to the inspection, seizure, and condemnation of meat which is "unsound, unwholesome, or unfit for the food of man." Wisely or unwisely, no attempt has been made in any of these enactments to define the conditions of meat or other animal products to which these adjectives may be applied, and the proceedings in our law courts have often proved that very different opinions with regard to the importance of particular diseases and abnormalities in this connection are held by different members of the medical and veterinary professions. In many of these cases in which disputes arise as to the unfitness of meat for human use the diversity of opinion expressed by the witnesses on the two sides cannot be said to be discreditable to either, since views may differ as to the facts and inferences even when all are honest and reasonably well informed. Unfortunately, however, there are other cases in which the opinion expressed by some of the witnesses can only be ascribed to a discreditable degree of ignorance or a reprehensible lack of the sense of responsibility in dealing with a question which touches human health.

At the present time this unjustifiable diversity of opinion is perhaps most frequently illustrated in prosecutions with regard to the sale of the flesh of animals that have died a natural death or been killed when already moribund. Not only the published proceedings of our law courts, but also other sources of information, from time to time bring under our notice the fact that too many veterinary surgeons hold on this matter opinions which are not creditable to their knowledge of pathology, and which when they are publicly expressed are calculated to seriously prejudice the claims of the veterinary profession to be considered the best judges as to the fitness or unfitness of meat for human food.

Quite recently a veterinary surgeon, recognising the hopeless condition of a cow he was called in to treat, advised the owner to kill the animal in order to save its carcase. The advice was followed, but, fortunately, the carcase was not saved, as the animal was the subject of anthrax and the fact was detected in the slaughter-house.

In justification of the advice which was offered to the owner of the cow in this case, it might be said that the veterinary surgeon in giving it took no actual responsibility as to the fitness of the carcase for human food, but assumed that if it should turn out to be unfit it was the duty of some other person to condemn it. In our view, however, this excuse is entirely inadmissible, for the simple reason that when an animal is already in a moribund condition from any cause its carcase, *ipso facto*, must beforehand be pronounced unfit for food. The only possible exceptions to this rule are cases of impending death from the gross mechanical injuries that come under the head of accident. It is quite possible that many veterinary surgeons who would readily assert to the view that the advice offered in the above-mentioned case was improper would nevertheless demur to the statement that the carcases of diseased animals slaughtered because their condition is considered hopeless ought always to be condemned, and it may be admitted that in certain circumstances the public health might be safeguarded without thus in advance declaring all such carcases to be dangerous. But these circumstances are not in existence in this country, where there is no obligation to report the fact when a dying animal is killed, and no surety that the *post-mortem* inspection will be conducted with the special care necessary to determine whether the consumption of the flesh by human beings would be devoid of danger. In this country thousands of carcases are never inspected at all, and many more receive only the perfunctory examination which is inadequate for the detection of dangerous conditions in the absence of the internal organs and of all history as to what was the animal's state immediately prior to slaughter.

If anyone is in doubt as to the unwisdom of advising the owner of a dying animal to have it killed and dressed for the market, he would do well to make himself acquainted with the history of cases of so-called meat poisoning in this and other countries. That history is mainly a record of deadly effects wrought on human beings by the flesh of animals slaughtered during the final stage of some obscure disease. The lesson which it teaches is so obvious that the veterinary surgeon who counsels an owner to save his pocket by cutting the throat of a dying or dead animal and putting the carcase on the market must be adjudged guilty of conduct which is little short of disgraceful. On this matter the veterinary profession ought to take a firm and united stand, and consistently refuse their professional sanction to a practice fraught with so much danger to the public.

CLINICAL ARTICLES.

A CASE OF PERITYPHLITIS IN THE DOG.

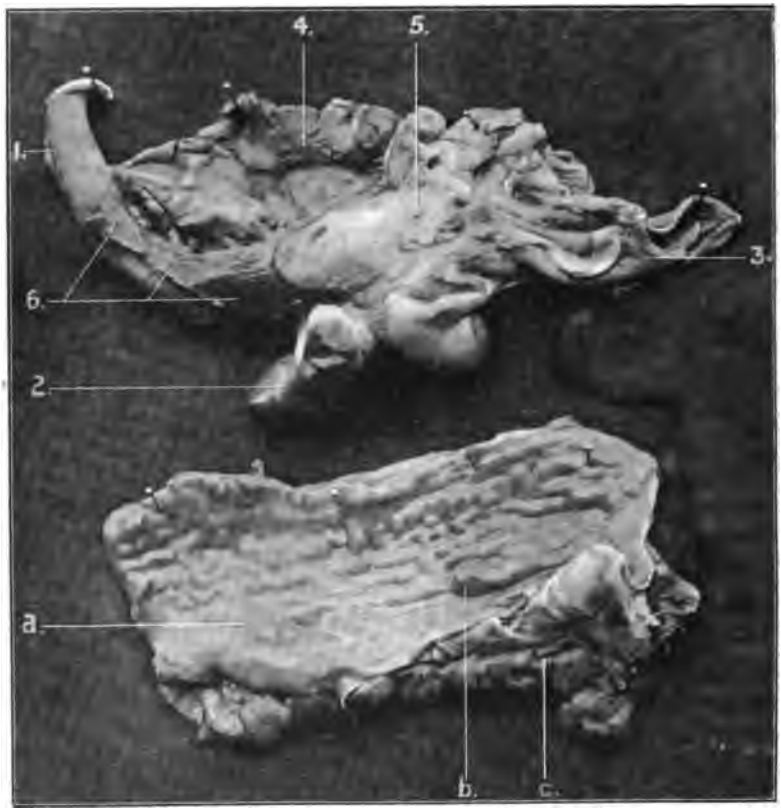
By GEOFFREY H. LIVESEY, M.R.C.V.S., Hove, Sussex.

AN old mastiff dog aged about eleven or twelve years had been unwell for the last seven or eight weeks, that is, his appetite and spirits had been variable and his bowels irregular. During the last three days he became dull and ate practically no food, though he was always thirsty. His attendant eventually came to me with a history of all the symptoms of canine typhus. I visited the dog, found him vomiting and in a collapsed state, and from a cursory examination made up my mind that he was suffering from typhus. I had him brought in and placed in an isolation kennel. Here I examined him very thoroughly. The temperature was 103°, pulse 146°, thin and rather thready, the skin tense and inelastic, extremities cold, and abdominal muscles rather contracted. The breathing was not fast, but each inspiration was effected with a distinctly separate thoracic and abdominal movement. The urine was passed on his bed, apparently involuntarily, and was dark in colour. He was able to stand and walk slowly, and the hind legs were not unduly stiff, nor was the gait straddling. Gentle manipulation of the abdomen produced no evident pain, and no tender areas could be located. The same morning he had received from his attendant 1½ oz. castor oil.

The following day he was semi-comatose, his temperature was one degree down, and his pulse very weak and rapid. Believing I had a case of typhus of some duration to deal with, I opened the saphena vein and transfused 7 oz. of (sterile) normal saline solution with 4 grs. of acetozone (Parke, Davis & Co.). The heart beats were at once much stronger, and the pulse much better in character and at the rate of 122 per minute. The dog seemed brighter and walked voluntarily to his bed, and even took some interest in the preparation of his food, though he could not be induced to touch any. The following day he was curled up comfortably when I saw him, breathing easily but very slowly. Temperature 102·6°. The skin was more elastic and the abdomen less tense, but in spite of these appearances, which I considered more hopeful, he was only with difficulty roused, and would take no interest in anything. He was given liquid food, and, as the bowels had not acted, the rectum was emptied with a glycerine and water enema.

The next morning I found him dead, lying stretched out to his full extent on his left side. *Post-mortem* examination was made without delay and revealed the following conditions:—

The abdominal cavity contained a large amount of blood-stained fluid, with small flecks of lymph in it, and having a peculiarly objectionable fæcal odour. I could not guess the exact quantity, as the contents of the bladder escaped into the cavity owing to a slip of the knife. The peritoneum and parts of the bowel and mesentery were ecchymosed, and nearly all the mesenteric glands were much enlarged (ecchymoses were also seen on the pleura). The stomach was contracted and empty, and showed much thickening of the walls; the mucous membrane was swollen and showed many small confluent hæmorrhages. The duodenum was empty and had several well-developed ulcers upon it, two at least having necrosed centres and raised edges. To the last four inches of the ileum and to part of the colon the mesentery was adherent. The distal end (vermiform appendix) was also adherent to the ileum. Of these adhesions the former was evidently quite recent, while the latter was of long standing. All the bowel here was of a black greenish colour, except for one or two dark purplish hæmorrhagic patches. The surrounding tissue was much thickened and fibrous. On breaking down these adhesions between the ileum and the mesentery, a small hole was exposed on the ileum about one-sixteenth inch in size, through which



Upper Specimen.—1. Ileum; 2. Cæcum (appendix); 3. Colon; 4. Omentum; 5. Opening of abscess; 6. Broken-down adhesions.
Lower Specimen.—Duodenum (opened); a and b, Necrotic ulcers; c, Pancreas.

a little thin and fæcal-stained pus escaped. This part of the ileum seemed to have some substance impacted into it; also the cæcum, the walls of which were thinner than normal, and whose distal end was distended with gas. When opened these parts contained what appeared to be ordinary fæces. This was washed, and proved to be ingesta and straw impacted round two small pieces of bone, which from their worn appearance I should judge had been lodged there for some considerable time. The remainder of the bowel was empty and showed the usual signs of typhus.

The above case presents some very interesting features. From the first I had made up my mind as to its being a case of typhus, though I thought the temperature was unusually high. I expected that if I found abdominal pain anywhere it would be in the region of the stomach and liver; but there was no evidence of any localised pain. There was no external discoloration of the skin. There was no fæcal odour in the mouth, nor more discoloration of the tongue than is generally found in typhus. Moreover, there was nothing in his behaviour or movement to lead one to suppose that there was any peritonitis present.

My view of the case, after making the *post-mortem*, is that the dog some ten weeks previously must have suffered from an adhesive perityphlitis and recovered spontaneously. This had been followed some seven or eight weeks later by an attack of typhus, which was taking a slow course, though ulceration was proceeding in the upper bowel. His constitution and digestion being much weakened, the bones he had eaten were not digested and became impacted at the seat of the old trouble, where no doubt the bowel was less elastic owing to the contraction of the newly-formed fibrous tissue. Here the inflammation set up ran on to suppuration, the abscess breaking into the abdominal cavity and causing a generalised peritonitis and death about the fifth or sixth day of the illness. Very possibly the latest formed adhesions, nature's remedy in such cases, were partially broken down by my manual examination of the abdomen.

I do not see what symptoms there are in this case to lead one to diagnose perityphlitis, but in future I shall watch more carefully those cases of suspected typhus in which there is an unusually high temperature.

I have three times previously found an inflamed appendix in the dog on *post-mortem* examination, and have had one case of an abscess penetrating the right rectus muscle, which I opened and drained, but I do not know if it had any connection with the bowel. I should think it unlikely, from the anatomical position of the cæcum, that an abscess forming there would burrow outwards to the skin. Probably in nearly all cases it would break as it did in the above case, and cause fatal septic peritonitis.

In contributing this paper I do so in the hope that it may help others to diagnose such a case during life in sufficient time to allow of the performance of laparotomy.

THROMBOSIS IN THE HORSE.

By S. I. JOHNSTON, M.R.C.V.S., attached Army Veterinary Department, Potchefstroom, Transvaal.

THE following two cases came under my notice lately whilst in veterinary charge of the 7th (Q.O.) Hussars at Potchefstroom, Transvaal.

Case I.—A big, underbred North American chestnut gelding, nine years old, used as a charger for the last two years without having had any serious ailment during that period.

He had been ridden in the ranks since July 1904, doing well up to September last, when twice within ten days he was sent in from field operations with a history that during a gallop or any prolonged fast work he would suddenly become very much distressed—blowing, sweating profusely, snatching up the near hind leg, and holding it up as if cramped. After standing a few minutes the symptoms would pass off, and the animal was able to walk back to barracks apparently quite well.

I saw him on each occasion when he returned, and could not detect anything amiss, but after the second attack he had a dose of aloes and a few days' rest. On being returned to duty I saw him do his first fast work, and there was a repetition of the former symptoms. After he was trotted for five minutes the man had to dismount very hurriedly, or the horse would have fallen down. During the attack the animal sweated profusely from the head to the lumbar region, while the quarters and thighs were dry.

I was convinced that it was a case of thrombosis of the vessels supplying the hind limbs, though, on making a rectal examination, I could not detect anything abnormal in these vessels. I sent the case to No. 3 Station Veterinary Hospital to be treated by Lieut. Plunkett, A.V.D., who put him on a course of potassium iodide and applied a blister over the loins, but without any beneficial results. A recurrence of the symptoms could be brought about at any time on exercising him at the trot for from five to ten minutes, when he would go down, to regain his feet after a few minutes' rest. These symptoms never occurred in the stable, nor at a walk. As the horse was no better after two months' treatment, he was destroyed.

A *post-mortem* examination, made in conjunction with Lieut. Plunkett, showed the abdominal organs to be healthy, also the lungs. The heart was very much hypertrophied on the left side. The posterior aorta for its last two inches was firmly plugged, the clot extending the whole length of both external and internal iliacs on either side, and for about half the length of the femoral on the left side. The clot was firmly adherent to the vessel walls for the greater part of its length.

Case II.—This was also a big, underbred North American bay gelding, eleven years old, and used as a charger. He was transferred to this regiment in September 1903 with a clean history sheet. About May 1904, when at work, he occasionally became affected with cramps in the hind limbs, which were attacked alternately. At first these would pass off, to recur in a few days with greater severity.

From its fleeting nature I thought the disease was of rheumatic origin, and sent him to hospital, where Lieut. Plunkett treated him up to the end of August, for rheumatic arthritis of the stifle, with varying success. He was then discharged to duty, the lameness having disappeared, though the spasmodic action remained in both hind legs.

The owner being on leave, the horse only did exercise work, the groom reporting that he went fairly well. On the night of the 19th November it was reported to the orderly farrier that he was suffering from spasmodic colic, for which he administered the usual aloes drink, and the horse very soon got relief.

On the evening of the 21st November I was informed that he had another attack. I found him standing (he had been rolling), with sweat streaming off him everywhere, very cold all over, breathing hurried, pale mucous membranes, the pulse very full and sluggish, temperature 99·4°. He was holding up the off hind leg as if suffering from cramp. I tested him for sensory paralysis, and got a normal reaction. As he was rather dangerous, a rectal exploration was not made.

The case was sent to hospital, where he remained without any further attacks up to the 30th November, when, in conjunction with Lieut. Plunkett, I had the horse exercised. He had only been ridden at the trot for five minutes when he got very distressed, blowing, with head extended, crouching of the hind quarters, knuckling over and dragging along on the hind fetlocks, and finally going down. As he was unable to regain his feet, we had him destroyed.

Post-mortem Examination.—The posterior aorta and external and internal iliacs were filled with an enormous clot, of a firm consistency and adherent to the arterial walls. There was well marked hypertrophy of the heart, with epicarditis and endocarditis. The lungs showed numerous inflammatory areas, some of which contained miliary nodules. The liver was also studded with miliary nodules. Other organs healthy.

Differential Notes.—No. I. (*a*) Always went sound until put to fast work, the period before attack varying from five to ten minutes. (*b*) He always sweated profusely from the neck to the loins, but not over the quarters.

No. II. (*a*) For the last four or five months of life the animal, if not actually lame, always had exaggerated action of one or both hind limbs. (*b*) He had two attacks in the stable (he had not done any work for at least six hours previously), each time sweating all over, while during the last attack the sweating was confined to the body, and not profuse.

TWO CASES OF TRYPANOSOMIASIS.

By ADAM GIBSON, M.R.C.V.S., Colonial Veterinary Surgeon,
Hong-Kong.

The first case was found in a Chinese buffalo. The animal was brought into the abattoir in the usual way. It was examined, and appeared to be quite healthy and in good condition. Next morning it was found dead. Blood films were at once prepared from the

Before destruction I made drawings of the situations of the lesions, which are here reproduced, and give an idea of the wide area over which they were scattered.

Post-mortem examination showed all the external organs healthy. On stripping the skin in the affected regions, it was found that the eruptions and cording were confined to the deep layers of the skin, and did not extend into the subcutaneous tissues. The cording was about the thickness of thin whipcord, and was very well defined.

The special point about this case is the simultaneous and widespread nature of the lesions, which is rather opposed to the generally accepted view that infection always takes place through some previous wound in the neighbourhood of the lesion.

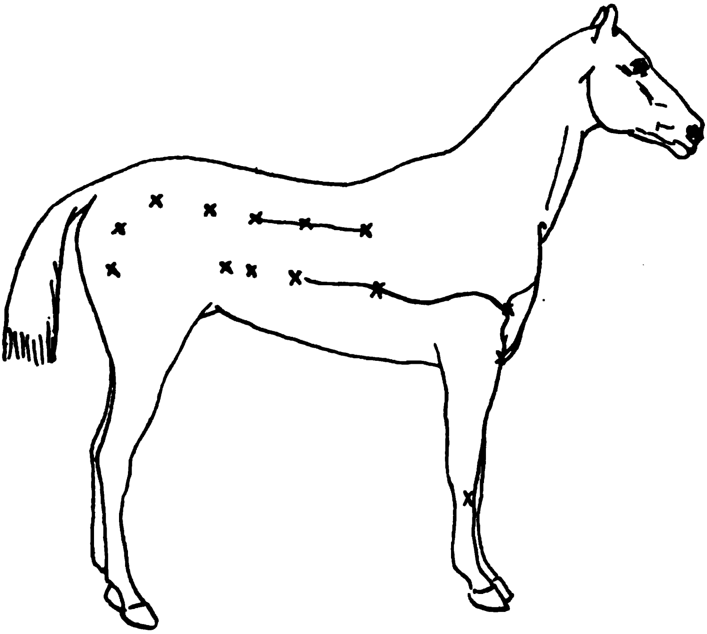


FIG. 2.

To indicate the distribution of the lesions on the off side.

One possible explanation occurs to me—viz., that there may have been some old infection which had remained in the form of an abscess, and that this had suddenly broken down and discharged the organisms into the circulation. Of course this is pure conjecture, and I could find no old lesion to support the theory.

I may add that the microscopical diagnosis was verified by several independent observers.

SUPPURATION OF THE SUBMAXILLARY LYMPHATIC GLAND IN GLANDERS.

By SYDNEY DODD, M.R.C.V.S., Demonstrator of Pathology,
Royal Veterinary College, London.

ALTHOUGH enlargement of the submaxillary lymphatic glands is a common symptom in clinical cases of glanders in the horse, yet actual suppuration of these glands is very rare. The following case shows that such a condition does occur, and, although it did not present any difficulty for diagnosis on account of other clinical symptoms, it indicates that the possibility of a discharging submaxillary gland being glanderous should not be lost sight of.

The subject, a brown mare aged seven years, was brought to the Out-patient Department of the Royal Veterinary College for treatment. Its general condition was fairly good. The off hind leg was thickened, and showed three ulcers on the inside of the fetlock and two on the inside of the hock. Inside and just below the stifle of the near hind leg was a small abscess, discharging a dirty yellow tenacious fluid. On the lower surface of the body, at the posterior third of the sternum, was a nodule about the size of a walnut.

The right submaxillary lymphatic gland was enlarged and indurated, the swelling extending to the outer surface of the angle of the right inferior maxilla. The skin over the gland had ruptured, and a thick viscid pus was being discharged, a good deal of which had dried around the opening.

No ulcers could be observed in the nostrils.

The animal was tested with mallein, reacted, and was destroyed.

On *post-mortem* examination both lungs were found to contain numerous nodules, varying in size from a split pea to a large cherry. The smaller ones were quite shotty, but some of the larger nodules were actual abscesses.

The bronchial and mediastinal glands were enlarged, and on section revealed several purulent areas. The Schneiderian mucous membrane was congested, but free from ulcers. The liver contained several echinococcus cysts, some calcified and others with thickened walls, and containing the usual clear fluid. Other organs normal.

Potato tubes inoculated from the sternal bud and from the lungs gave a pure primary growth of glanders bacilli, while one inoculated from the submaxillary gland yielded a mixed growth of glanders bacilli and other organisms, no doubt owing to external contamination of the abscess.

Abstracts.

THE DIAGNOSIS OF MAMMARY TUBERCULOSIS.

OSTERTAG, Breidert, Kaesewurm, and Krautstrunk undertook an investigation for the purpose of defining the symptoms peculiar to the different stages of mammary tuberculosis and the reliability of the various recently suggested means of diagnosis· viz., examination of the udder, detection of tubercle bacilli in stained smear-preparations of the milk, inoculation of experimental animals, and "harpoonage" of the udder. Their conclusions were as follows:—

(1) Tuberculosis of the udder is clinically indicated by swelling and hardening of one or more quarters. As stated by Bang and Moser, the disease usually begins in one of the posterior quarters and takes a chronic course. The swelling causes the teats of one half to assume a parallel or converging direction, instead of diverging from one another. A healthy quarter feels soft or moderately firm throughout; in milch cows the normal elastic tissue often contains symmetrical firm masses of varying size formed by the lobules of the gland. A tuberculous quarter feels irregular from the beginning of the disease. Certain portions, especially towards the back of the udder, and over the milk cisterns, are firm, stiff, or as hard as wood. The firm parts form more or less sharply margined nodules, distinct from the normal elastic tissue. The tuberculous swellings increase in circumference, and in time their surface becomes nodulated.

When the udder is distended the tuberculous swellings are often difficult to detect, but in a relaxed state this difficulty disappears. The swellings are painless and of the same temperature as their surroundings.

As shown by Bang, Müller, Lindemann, and Langer, the milk of the diseased quarter may remain of apparently normal constitution for weeks. As the tuberculous process extends and destroys the secreting tissue, it changes in appearance, becoming thin, poor in fat, flocculent, and finally watery. At this time it often exhibits an alkaline reaction instead of the normal neutral reaction.

In exceptional cases tuberculosis of the udder may commence with acute inflammation, or may for a time assume an acute course. The lymphatic glands of the diseased quarters or half are always swollen. Their condition can most conveniently be examined by thrusting the skin covering the side of the diseased hind quarter upwards, towards the flank, with the index, middle, and ring fingers, and palpating the posterior and lateral aspects of the glands in question. When diseased the glands are found to be enlarged in all directions; sometimes their surface is nodulated.

Kühnau has shown that disease simulating tuberculosis of the udder may be produced by chronic infection with streptococci or with actinomyces. In the chronic inflammation due to streptococci the lymph glands are greatly enlarged but are not nodulated. In actinomycosis of the udder the lymph glands are seldom much enlarged.

(2) The clinical diagnosis of tuberculosis of the udder may be regarded as assured when one quarter of the udder and its attached lymph glands exhibit firm, hard, nodulated swellings without signs of inflammation. When the quarter and attached lymph glands exhibit only firm, hard swellings, without nodulation, the diagnosis is somewhat doubtful. Suspicion, however, will be increased if the milk from the suspected quarter is of apparently normal constitution, or appears, from the history, to have been of normal constitution at the commencement of the disease. The probability of

tuberculosis is again increased by the existence of any other clinical indications of the disease, such as (1) general falling off in condition; (2) continued fever without apparent acute disease; (3) painless, nodulated swellings of the pharyngeal, prescapular, or precrural lymphatic glands without symptoms of inflammation; (4) frequent. spontaneous, weak cough, and pneumonic râles without apparent acute inflammation; (5) frequently recurring tympanites without apparent cause; (6) frequent œstrum and muco-purulent discharge from the vagina. In rare cases symptoms of tuberculosis of the brain and tuberculosis of the vagina may lend additional weight to the suspicion of tuberculosis of the udder.

Portions of the prescapular and precrural glands may be removed without danger. Removal of portions of the mammary lymph glands is somewhat difficult on account of the deeper position of these parts, but is also without danger. Macroscopic examination of such fragments is often sufficient to reveal the tuberculous nature of the disease.

To detect the pneumonic râles it is best to trot the animals. Pilocarpin and arecolin have been suggested as likely to prove useful in the diagnosis of tuberculosis of the lungs, but their value can only be established by further experiment.

(3) Contrary to Dr Rabinowitsch's statement, the tuberculin test is not of practical value in the detection of tuberculosis of the udder. In the first place, it may fail; but, apart from this, the occurrence of a reaction does not prove that a suspicious swelling in the udder is tuberculous, for the reaction may be due to a tuberculous lesion in some other part, such as the bronchial, mediastinal, or mesenteric lymphatic glands, a lesion which is unimportant as regards the continued use of the animal or the spread of tuberculosis.

(4) As a rule bacteriological examination is indispensable for the conclusive diagnosis of tuberculosis of the udder. Of the various forms of bacteriological examination undertaken with this object, inoculation of guinea-pigs with suspected milk is the most reliable, as shown by L. Rabinowitsch and O. Müller. One cubic centimetre of milk as withdrawn is sufficient. The best method is to inject the milk into the muscular substance of the inner and posterior surface of the hind limb. This is as reliable as intra-peritoneal injection, which was formerly the most widely employed, and was regarded by Rabinowitsch as the most conclusive, while it has the advantage of being much speedier. The experimental animal can be killed for further examination as soon as the lymph glands near the point of inoculation appear firm, hard, painless, and enlarged to the size of a small pea. This often occurs within ten days of inoculation, but should the lymphatic glands not become diseased the experimental animals are killed six weeks after inoculation. The discovery of tubercle bacilli in the enlarged lymph glands or internal organs confirms the diagnosis. Intra-muscular injection obviates sources of error due to the pseudo-tuberculous changes which so often follow intra-peritoneal injection with milk accidentally containing acid-fast pseudo-tubercle bacilli. The entrance of acid-fast bacilli (which, by the way, can usually be recognised as such on account of their shape) can be avoided, if. before withdrawing the milk, the udder be washed with soap and water, cleansed with 50 per cent. alcohol, and rubbed dry with sterilised wadding. The first 10 cc. at least of the milk should be thrown away. Finally, the intra-muscular method has the great practical advantage over the intra-peritoneal that much fewer experimental animals die from intercurrent diseases.

Examination of the milk by means of smear preparations and bacterioscopic examination of harpooned fragments of the tissue of the udder are uncertain. Despite the existence of the disease a considerable number of such tests may fail. This conclusion, at least as regards smear preparations, is in harmony with the reports of Rabinowitsch and Müller. The last method of examination is only conclusive when it yields a positive result. Nevertheless, as stated by Bang and Müller, it is valuable for the rapid diagnosis of advanced tuberculosis of the udder. In certain exceptional cases harpooning of the udder may prove more reliable than inoculation. For bacteriological examination the milk should be passed through a centrifugal separator and the precipitate used.

Harpooning necessitates casting the animal. By carefully disinfecting the skin and using sterilised instruments, the operation is rendered harmless even if repeated several times. The skin and fascia of the udder are first divided, the suspected portion of the udder grasped with the hand, the harpoon introduced into the supposed diseased part, rotated slightly, and rapidly withdrawn. A small fragment of tissue is sufficient, provided it contains one or two tubercles the size of a millet seed. Harpooning may be resorted to when inoculation has failed to confirm an otherwise strong suspicion of tuberculosis, or when milk has ceased to be secreted and therefore cannot be employed.

(5) In advanced cases of tuberculosis of the udder milk from the diseased quarter may be virulent even when diluted to the extent of one to one billion. In commencing tuberculosis of the udder, and sometimes in advanced cases, the number of bacilli is very small, and the virulence of the milk can be destroyed by dilution to a greater degree than 1 to 1000.

(6) In examining nasal, pharyngeal, and vaginal discharges for tubercle bacilli, it must be borne in mind that these materials, like the fæces, contain acid-fast pseudo-tubercle bacilli, and therefore that in doubtful cases the results must be checked by inoculation experiments (*Zeitschr. f. Fleisch und Milch Hyg.*, 1905, p. 1, and *Deutsche Tier. Wochens.*, 7th January 1905).

IMMUNISATION AGAINST TUBERCULOSIS BY VON BEHRING'S METHOD.

In November 1902 Dr von Behring sent two oxen protected by his method from Marburg to the University at Leipsic for the purpose of being tested as to their degree of resistance against artificial infection with bovine tuberculous material. The tests were carried out during the autumn of 1904. Ox No. 1 had received intravenous injections of bovine tubercle bacilli, the activity of which had been diminished by treatment with trichloride of iodine, followed by intravenous injections of bovine tubercle bacilli dried *in vacuo*, and an injection into the anterior chamber of the eye of virulent human tubercle bacilli. The eye became tuberculous and was removed by operation. Finally, the animal was subjected to a series of six injections with increasing quantities of fresh human tubercle bacilli.

Ox No. 2 had previously been injected with dead human tubercle bacilli cultivated in the rat and with fresh human tubercle bacilli. Following on this, it had received an intravenous injection of fresh bovine tubercle bacilli, and finally had been twice inoculated intravenously with avian tubercle bacilli dried *in vacuo.*

Oxen Nos. 1 and 2 were then gradually infected, both subcutaneously and intravenously, with bovine tubercle material, six other recently bought and tubercle-free young oxen being employed as controls. Two other freshly bought tubercle-free young oxen were utilised for the purpose of controlling the method of feeding and the general hygienic conditions.

The general conclusion arrived at was that the oxen treated by the Marburg method showed a greater degree of resistance against artificial, subcutaneous, and intravenous infection with tubercle virus than those not so treated. The grounds for this conclusion were:—

(1). The absence of any local change at the point of inoculation in oxen

Nos. 1 and 2 when infected with slightly-virulent material, which in control ox No. 6 produced tuberculous infiltration at the point of inoculation and tuberculous enlargement and caseation of the neighbouring lymph glands.

(2). The very trifling local changes at the point of inoculation and the absence of any tuberculous change whatever in the local lymph glands in ox No. 1 when subcutaneously inoculated with very virulent material, which caused in control ox No. 13 extensive infiltration and ulceration at the point of inoculation, extensive swelling and caseation of the neighbouring lymph glands, and embolic tuberculosis of the lungs, liver, and spleen.

(3). The absence of any kind of tuberculous change in neighbouring lymphatic glands even in cases where subcutaneous injection of very virulent material led to the formation of a caseous tuberculous abscess at the point of inoculation and isolated embolic tubercles in the kidneys (ox No. 2).

In control ox No. 14 the subcutaneous injection of similar material produced extensive tuberculous infiltration at the point of inoculation, extensive tuberculous swelling and caseation of neighbouring lymph glands, and embolic tuberculosis of the lungs and spleen.

(4). When virulent tubercle bacilli were injected into the veins the increased resistance was principally shown by the trifling degree of general disturbance produced and by the speedy and substantial improvement in the condition of the protected ox, which continued to live for five months and three weeks longer. The animal was eventually slaughtered on account of acute brain disease, probably of tuberculous origin. Control oxen Nos. 22 and 21 injected with a similar amount of virulent material died in twenty-eight and thirty-eight days respectively.

When intravenously injected with attenuated tubercle material the protected and unprotected animals exhibited less difference in their powers of resistance; for even control ox No. 3 only showed trifling disturbance, and on *post-mortem* examination only slight tuberculous lesions.

The two protected animals showed different powers of resistance, No. 1 being more resistant than No. 2.

It is impossible to say with certainty how far this difference was due to the methods employed for the protection of the animals, and how far to the general weakening of the latter animal's constitution as a result of pregnancy.

The protected animals were by no means absolutely proof against the disease. When the quantity administered was sufficient both animals suffered from tuberculous infection.

The tuberculin test is unreliable in cattle previously treated with attenuated bovine or human tubercle bacilli unless a long period (not less than six months) has elapsed since the last infectious material was injected. The protected animals did not react to tuberculin even when they were suffering from tuberculous changes both at the point of inoculation and in their internal organs. As neither of the protected animals was immunised by the double inoculation method with weakened human tubercle bacilli according to von Behring's present method, the foregoing conclusions must not be applied to this method.

The results of the experiments show, however, that it is possible to confer a certain degree of resistance to artificial infection with tuberculous material by previously treating the animals with attenuated bovine or human tubercle bacilli.

Whether the more recent method of twice-repeated inoculation with attenuated human tubercle bacilli will increase the powers of resistance to such a degree as to afford protection against natural infection can only be ascertained by careful observation for several years of large numbers of animals so protected, as well as control animals. The results would require to be confirmed by *post-mortem* examination.

Numerous experiments bearing on this point are being made, partly in the

Veterinary Institute of the University of Leipsic, partly on farms. The conclusions will be reported later.

Eber considers the results so far obtained as very encouraging. (Eber, *Berlin. Tierärztl. Wochens.*, 1904, p. 888).

THE EFFECT PRODUCED ON THE VIRULENCE OF CULTURES OF THE SWINE ERYSIPELAS BACILLUS BY ADDING SERUM OR GRAPE SUGAR TO THE NUTRIENT BOUILLON.

In preparing cultures of swine erysipelas it was found that, whether the bouillon was prepared from meat or from extract, with the addition of peptone and common salt, the bacilli often failed to grow freely. It therefore seemed interesting to enquire whether greater freedom of growth without loss of virulence could be effected by making certain additions. Previous experiments had shown that the addition of 1 to 2 per cent. of serum or grape sugar to the bouillon caused the cultures to grow vigorously. In the first case, where serum was added, it was often impossible, despite repeated filtering, to obtain a clear fluid.

In the second instance, where grape sugar was employed, the fluid necessarily was clear, but soon after inoculation it became very turbid and in time deposited a greyish-white precipitate.

Small quantities, such as ·1 per cent. of serum and ·2 per cent. of grape sugar, encouraged growth equally well. In both cases the culture was at first clear. When serum was added the liquid remained for a time untroubled, but gradually a greyish-white precipitate formed which could, by agitating the liquid, be rolled up into fine clouds. Soon after the grape-sugar-bouillon was inoculated marked turbidity developed.

The bouillon used in the following experiments was carefully neutralised; for the first three days the cultures were placed in the incubator at 37° C., afterwards they were kept at the ordinary temperature of the room.

White mice, which, as is well known, are very sensitive to swine erysipelas, were used as tests. In each case ·5 cc. was injected into the peritoneal cavity.

The first series of experiments was for the purpose of determining whether the addition of ·1 per cent. of serum to the bouillon caused changes in virulence, and how long the cultures so obtained remained active.

For some time before death the animals sit motionless, with the eyes closed and the eyelids glued together by discharge; they often die in this position. All the experimental animals died within three to seven days after injection. In most cases the liver, spleen, and kidneys were much enlarged. The bacilli were usually found isolated, but were in greatest number in the leucocytes.

The addition of ·1 per cent. of serum had therefore certainly not diminished the virulence of the cultures, for six-weeks-old serum-cultures were still capable of killing experimental animals in four days. Cocci derived from the air and spore-forming bacilli, which sometimes obtain entrance to the cultures, do not appear to diminish the activity of swine-erysipelas bacilli when cultivated in serum bouillon, a fact of some importance in relation to the practical use of such cultures on farms, etc.

It is known that many bacilli when grown in 1 to 2 per cent. solutions of grape sugar gradually cause the formation of acids, and that the acidity of the culture interferes with the growth of the bacteria and with their virulence. Previous experiments had shown, however, that the bacilli of swine erysipelas remained alive for four weeks in bouillon containing ·2 per cent. of grape sugar, and it therefore seemed worth while testing the virulence of such cultures.

A two-days-old culture of swine erysipelas bacilli in bouillon containing 2 per cent. grape sugar was of neutral reaction; traces of grape sugar could still be detected chemically. The activity of such a culture had not suffered, and even a four days' culture still killed mice in seven days. The latter culture, however, showed considerable chemical change. Grape sugar could no longer be detected, and the solution had become acid. As the formation of acid proceeded the bacilli became markedly affected, and the injection of such cultures produced no effect.

The experiments proved that as the acidity increases the virulence diminishes. Mice injected with fourteen and thirty days' old cultures did not show the slightest sign of disease even after the lapse of twenty days. As the bacilli in such cultures still remained alive even after thirty days, it seemed possible that they might be used for the purpose of conferring on experimental animals a certain degree of immunity against swine erysipelas.

The object of the experiments was to discover whether mice injected with distinctly acid grape sugar cultures still remained sensitive to the injection of virulent cultures produced without the addition to the bouillon of serum or grape sugar.

The experiments. however, failed to produce any degree of immunity against swine erysipelas. The decomposition produced by the life activities of the bacilli rendered the cultures completely impotent. During the course of these experiments it was also found that ordinary bouillon cultures, without any addition, retained their activity even when containing cocci derived from the air.

The general conclusions arrived at may be stated as follows:—

The addition of ·1 per cent. of sheep's serum to bouillon increases the growing powers of the bacilli of swine erysipelas. Such cultures, like ordinary bouillon cultures, do not lose their activity even after six weeks.

Contamination of the cultures with air-borne cocci and spore-bearing bacilli apparently exercises no injurious effect on the virulence. The addition of even small quantities of grape sugar to the bouillon causes the bacilli at first to grow with extraordinary rapidity, but the grape sugar soon undergoes decomposition as a consequence of the life activity of the bacteria, and substances are produced in the acid bouillon which, though for a long time incapable of killing the bacilli, very rapidly destroy the virulence of the cultures (Gordan, *Berlin Tierärztl. Wochens.*, 1904, p. 891).

TRANSMISSION OF GLANDERS BY A HORSE WHICH HAD REACTED TO MALLEIN BUT EXTERNALLY APPEARED HEALTHY, AND RE-INFECTION OF TWO HORSES WHICH HAD CEASED TO REACT TO MALLEIN.

THE French Ministerial Circular of 15th September 1894 codifies the sanitary precautions applicable to horses suspected of glanders and which have reacted to mallein.

Those which show no clinical sign of the disease are to be placed under inspection for a year, the owner being allowed to use them under certain restrictions laid down by the Consultative Committee on epizoötic diseases.

These restrictions direct that animals exhibiting the ordinary reaction, *i.e.* fever, local swelling, prostration, etc., shall be regarded as suspected; they must be rigorously isolated from the others, marked, and kept under observation for a year in the same way as those which have shown some symptom due to glanders. During this period of observation they must be tested with mallein every two months, and kept under observation until such time as they shall have failed on two successive occasions to react to this test. They may be used provided they present no symptoms suggesting that they are dangerous, and provided they are not allowed to drink at common watering places or to enter any other than their own stable.

Though theoretically perfect these sanitary precautions do not produce the expected results, except where the animals can be rigorously isolated. In numerous cases space is insufficient, and horses which have reacted are placed in one part of the stable, isolated by a simple partition or gangway from those which have failed to react to mallein. From the commencement of the inspection period special utensils are kept for cleaning them and for containing their drinking water and food. Such vessels are disinfected after each meal in order to prevent danger from involuntary substitution. Somewhat later, however, as the period of inspection advances, these precautions are neglected. The owner, seeing the suspected animals apparently in good health, no longer believes in the reality of danger nor in the warnings furnished by the injection of mallein. He comes to regard the precautions as vexatious and useless. Although the fear of fine or imprisonment prevents him removing animals which have reacted to other stables, or watering them at common drinking troughs, or employing them under conditions other than those laid down by the municipal authorities, he ceases voluntarily to observe the prescribed measures called for in his own interest. In several cases this blind obstinacy has resulted in the appearance of glanders amongst horses which in the first instance had failed to react to mallein. Amongst others the following cases deserves to be recorded on account of the lesson it conveys.

Glanders was discovered in an establishment where, after slaughter of the clinically diseased animals, five horses were left which showed no external sign whatever of glanders. Of this number two failed to react and three reacted to mallein. The former, isolated in a corner of the stable and then removed to another place, never reacted. Those which showed a reaction were working in a public vehicle and lived together, eating from the same manger.

The second test, made two months later, gave the following results as regards the animals of the second series: complete reaction, *i.e.* fever, local swelling, prostration etc., in one; trifling reaction in the second; and no reaction whatever in the third. Despite the advice given him, the owner did not think himself bound strictly to isolate each of his horses. Forty-two days after the second injection of mallein the animal which had not reacted showed glanders in the nose. On *post-mortem* examination other lesions of sub-acute glanders were found in the Schneiderian membrane and lung. In spite of the most careful search no old lesions were detected.

On the second test the suspected horse again reacted, and its neighbour in the stable was declared healthy. Thirty-four days later the non-reacting horse showed signs of glanders and was slaughtered. On *post-mortem* examination glanders lesions, all of a recent character, were found in the mucous membrane of the nose and in the lung.

The suspected animal remained under observation for four months longer. Every two months it was re-tested, and on each occasion it reacted. Yielding at last to advice, the owner consented to its being slaughtered. The *post-mortem* examination revealed very old standing lesions of chronic glanders, a condition rarely met with at the present time, inasmuch as animals are usually destroyed early. All the organs were free from disease except the lung, which was dotted over with hard rounded nodules, varying in size between that of a pea and a small hazel nut, and consisting of a thick fibrous wall intimately connected with the pulmonary tissue and containing a central caseous mass of a whitish-yellow colour infiltrated with lime salts. The enlarged bronchial glands were hard and nodulated, and showed on section numerous centres which had undergone caseation.

This observation is interesting for several reasons. In the first place, it shows the dangerous character of horses which have reacted but are otherwise apparently healthy. The two horses which ceased to react to mallein were re-infected by their neighbour. This animal showed no clinical signs but gave a complete reaction after each test.

This fact entirely justifies the precautionary measures recommended by Nocard after his first investigation into the prevention of glanders. It also justifies a refusal to accept, save with very distinct reservations, the conclusion of the English Committee, that under ordinary conditions the danger of contagion by horses which have reacted to mallein, but clinically appear healthy, is so trifling that it may be neglected.[1]

The case conveys another lesson. It furnishes a further demonstration of the already recognised fact, viz., that in glanders a primary infection though successfully surmounted does not increase the powers of resistance either of the dog (Galtier) or of the horse (Nocard) to the glanders virus. The two horses next the glandered animal gave a complete reaction on the first test. They were therefore at that moment suffering from glanderous lesions. The healing of these lesions was shown by their failing to react to the second and third injections. Re-infection occurred after the second test in the first subject, and after the third test in the second. This case agrees with one recorded in the *Maladies Microbiennes.* Nocard obtained from a severely infected stud three horses which had several times reacted to mallein and afterwards ceased to react. These he fed with virulent cultures. The three animals which had recovered from glanders took the disease as readily as a control animal. He thus clinically proved that a first attack of glanders does not confer immunity (Conte, *Rev. Gén. de Méd. Vét.*, Vol. V., p. 199).

DR THEILER ON THE PIROPLASMA BIGEMINUM OF THE IMMUNE OX.[2]

THE piroplasma bigeminum is found in all cases of redwater during the fever stage and disappears afterwards. It also occurs in the blood of a recovered animal, although its form and shape have not hitherto been described. The strongest proof that the piroplasma is present in the blood of immune oxen is to inject such blood into young calves, when the typical piroplasma reappears, causing a reaction. This experiment has been made use of as a method of preventive inoculation, since it has been noticed that the reaction does not, as a rule, cause death, but produces immunity. For some time it has been known that after the first reaction. in some animals at least, a second occurs which may even prove fatal, and that during this reaction the piroplasma bigeminum reappears. Afterwards immunity is established. In an immune ox the usual form of piroplasma bigeminum may occasionally be found. If, however, the typical piroplasma, in its usual pear shape, is thought to be present in every immune ox, then the organism must be so rare as to escape microscopical examination. It is also believed that the parasite may change to some other state, in which it remains dormant.

When the examination of the blood of a calf which has been injected with blood from an ox immune against redwater is made and is continued after the second reaction has taken place, then a peculiar phenomenon is observed, namely, the occurrence of endoglobular parasites which correspond to the description of the piroplasma parvum, or piroplasm of East Coast fever. Sometimes they infest a considerable number of red corpuscles, but never as many as in cases of tropical piroplasmosis of some duration. Nevertheless, the microscope reveals a picture resembling that found in East Coast fever at the early stage of the disease, so that an error, unfortunately, is more than likely. Dr Theiler has observed rings and rods (bacillary forms) similar in size and shape to those described above. In some instances these organisms are already present during the second reaction after the inoculation of redwater-immune blood. Usually, however, they appear after the second reaction is over, and sometimes along with piroplasma bigeminum in its typical shape. Increasing for the first few days, they may in exceptional cases infest about 10 per cent. of red corpuscles, decreasing gradually. Some calves show them in varying numbers, for the first few months one to two in each microscopical field, but the decrease is maintained, so that after several months they have become so rare that it needs much patient searching and successful staining to find them. These parasites are still found in the blood of animals even after the lapse of a year.

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[1] No English Committee has ever arrived at such a conclusion.—EDS.

[2] "The Journal of the Royal Army Medical Corps," November 1904.

In several cases the appearance of these organisms is preceded or accompanied by poikylocytosis of the red corpuscles, and by the formation of basophile granulations and nucleated red cells. The basophile granulations are either uniformly distributed in the blood cells, and may be of different size, or appear only as a single dot, nearly always on the margin of the blood corpuscle. These granules cannot be mistaken for the above-mentioned organisms, since they are of a uniform dark blue when stained with methylene blue, and contain neither a chromatic body nor an achromatic zone.

Dr Theiler considers these organisms as the type of piroplasma bigeminum in the immune ox, which when injected into fresh susceptible animals reappear in the form of the pear-shaped parasites. This immune form has nothing to do with piroplasma parvum, with which it is often easily mistaken. The following experiments support this view:—

Experiment 1.—Ox VI., about two years old, born in Aliwal North, Cape Colony, from where it was imported, a district where no redwater is known. It was tied up in the station and never left outside the premises. On 30th July 1903 it was injected with 10 cc. defibrinated blood of Ox No. 347, which had contracted ordinary redwater towards the beginning of November 1902. Already on the fourth day after injection the temperature began to rise in Ox VI., reaching on the fifth day 106·2° F. On the next day the typical piroplasma bigeminum was present in rather large numbers. On 9th August 1903 red urine was avoided for the first time; on 13th August piroplasma bigeminum was still present, but the urine cleared up. On 14th August the first basophile granulated red corpuscles appeared, they increased during the next few days and a marked poikylocytosis made its appearance. On 16th August piroplasma bigeminum was rare; the temperature was then 104·2° F. in the evening. A week later the basic granulations were still noticed. On 9th September 1903 a second reaction set in, which lasted about eight days, and the evening temperature rose as high as 105·8° F. On 12th September 1903 the blood was examined and small endoglobular organisms resembling piroplasma parvum were found in the red corpuscles. The basic cells reappeared the following day. The parasites increased during the subsequent days, and poikylocytosis and nucleated corpuscles also appeared. On 15th September the parasites were in such numbers that the presence of tropical piroplasmosis was suspected, and the count revealed an infection of 10 per cent. of all corpuscles. On the 17th the number began to decrease, the basic cells being still abundant. Finally they almost entirely vanished; but whenever at any later date the blood was examined they were found. During the second reaction nothing appeared amiss with the ox.

In order to remove all doubt as to the possibility that this ox had contracted tropical piroplasmosis, it was determined to endeavour to infect it with that disease. On the assumption that it had contracted that disease, it ought now to have been immune.

On 15th February 1904, the blood of this ox was examined and the typical rings were still found present in small numbers. On this date the ox was infested with twenty ticks (Rhipicephalus appendiculatus). These ticks had been feeding as nymphæ on an ox suffering from East Coast fever. The same brood of ticks had already produced the disease in three other oxen. On 23rd February 1904, the temperature of Ox VI. began to rise, and kept up during the next thirteen days, and on 7th March Ox VI. died of tropical piroplasmosis. The piroplasma parvum was present in large numbers.

It may therefore be concluded that the two reactions in which the small endoglobular parasites were seen had nothing to do with one another; and that piroplasma parvum, although resembling in form and size the endoglobular parasite found in the second reaction of Ox VI., is altogether a different species.

If the immune form of piroplasma bigeminum is really only found after redwater reaction, then it follows that it will not be found in animals which are susceptible to this disease. This point can easily be settled by examining blood of freshly imported cattle from England, and of calves which are born in stables in the Transvaal. Such cattle, when injected with blood from an immune ox, should naturally show the appearance of rings and bacillary piroplasmata.

Experiment 2.—Calves 240 and 241 were both born on the premises of the laboratory; they were, at the time in question, still sucking, and there was no chance that they had previously contracted tick infection. Their blood was repeatedly examined, and found to be free of any endoglobular parasites. On 18th February 1904 the blood of the redwater-immune Ox 347 was examined, and the typical rings were found to be present in small numbers. The ox was bled, and the defibrinated blood was injected into the calves. Calf 240 was injected intraperitoneally, and Calf 241 subcutaneously, with 10 cc. defibrinated blood.

Calf 240.—The blood, which, after the injection, was daily examined, showed piroplasma bigeminum for the first time on 24th February. There was no rise of temperature. The next day the parasites were still present, also on the 26th, 27th, and 28th. On the last date a slight elevation of the morning temperature began, but there was never any distinct reaction. On 1st March the blood was found to be free of parasites. From 11th March a reaction began, during which the temperature rose considerably, reaching 106° F. on 17th March. From 13th March onwards the marginal basic points were now noticed in the red corpuscles; poikylocytosis and basophile red corpuscles appeared, and were present on 23rd March, when piroplasma bigeminum was again visible. From that date (23rd March 1904) onwards a few rings and bacillary forms were noticed; they were present the next day, and on the 28th had considerably increased. The piroplasma bigeminum was still found in preparations dated 26th and 27th March. The following days the rings still increased, and on 7th April they were so numerous that the microscopic field resembled an infection due to piroplasma parvum at the beginning of East Coast fever. There were sometimes two and three rings and rods in one corpuscle. A decrease took place from 13th April 1903. A daily examination was continued up to 1st June 1904, and rings and bacillary forms were constantly found.

Calf 241.—The temperature of this calf began to rise on 7th March. On 13th March there was a distinct poikylocytosis present, a few basophile red corpuscles, and also the basic marginal points, and these were daily noticed up to 26th March. On 21st March piroplasma bigeminum appeared, and the first bacillary forms and rings were noticed. After the 26th the rings were somewhat frequent during the days following. On 13th April basophile cells were still present. The decrease of rings began about 13th April. The blood of the animal was also daily examined during the next months, and up to the present time the rings are still present in small numbers.

When Calf 243 came into a second reaction it was deemed advisable to test whether the blood of this reaction would produce the same effect as the immune blood. Accordingly two calves, Nos. 242 and 243, were injected. These calves were also born on the premises. They served as controls for Nos. 240 and 241, and during this time never showed any endoglobular organisms. Both calves were injected with 10 cc. defibrinated blood under the skin on 17th March 1904.

Experiment 3.—Calf 242. On 29th March 1904, the first piroplasma bigeminum was noticed. There was no distinct temperature reaction. The parasites were seen again on 3rd April, from which date an irregular course of temperature began. On 17th April the piroplasma bigeminum was again found. On the 18th a few basophile marginal points were noticed in red corpuscles, and on the 19th the ring forms were already present. On 21st April rings and bacillary forms were seen in fair numbers. There were basophile granulations in red corpuscles. On 25th April basophile cells were still present, so also were the piroplasma bigeminum and the rings and bacillary forms. The piroplasma bigeminum disappeared again, the number of red cells with basophile granulations became reduced, and the rings remained in moderate numbers.

Calf 243.—From the sixth day after the injection of the blood a distinct reaction set in, lasting about seven days. The piroplasma bigeminum was observed on 26th March, when the temperature reached 106·4° F. On 25th March 1904, poikylocytosis made its appearance, the piroplasma bigeminum was present during the next five days, and then gradually disappeared. The poikylocytosis was noticeable during that time. On 19th April the poikylocytosis was again noticed, and also were the marginal points and the basophile granulations. The piroplasma bigeminum appeared on 20th April for the second time. The rings and bacillary forms appeared on the 26th, on which date a few basic cells were still noticed. After that date the bacillary forms and rings were continually found in the blood, but in gradually decreasing numbers.

The above experiments were made for the purpose of ascertaining whether, after an inoculation of immune redwater blood, the appearance of rings and bacillary forms is a necessary result. From various casual experiments made at a previous date, when the true nature of these rings was not yet apparent, the author had remarked endoglobular parasites identical with those just described, although in the latter case the examination of blood was not carried out from the very beginning. The oxen used in this experiment all came from Aliwal North, a country free of redwater. The idea of the experiment was to ascertain whether the blood of an immune redwater ox, mixed with serum of a hyperimmunised ox simultaneously injected, or serum injected before virus, or virus injected before serum, modifies in any way the reaction due to the immune redwater blood. Twelve animals were used for the experiment, two being controls, and the result showed that the serum had no influence on piroplasma bigeminum, inasmuch as the controls behaved exactly as the serum-treated animals. In some of the animals a disturbance of the temperature was noticed similar to that in ordinary redwater. whilst others showed no reaction.

The result of the inoculation was the appearance of rings and bacillary forms in the majority of the animals. It may therefore be concluded from the above experiments that the appearance of rings was due to the injection of immune redwater blood, the serum having had no effect whatever, the blood giving rise to reactions such as are usually observed in redwater inoculations. The examination of the blood was in some animals negative, which is not to say that the parasites were entirely absent, but merely that they escaped observation.

In addition to the evidence brought forward above, the author quotes the following: Eighteen imported English pure bred heifers, belonging to the Hereford, Shorthorn, Jersey, Lincoln, Polled Angus, and Aberdeen Angus breeds, were inoculated against redwater: two on 10th September, four on 24th October, ten on 14th November 1903. With the exception of two, all had reactions; most of them had primary reactions, during which time the piroplasma bigeminum was noticed; while in the majority of the heifers poikylocytosis and basophile granulations of the red corpuscles were observed. Four of the animals died, two from the primary reaction and two from the secondary reaction. Some weeks after the reaction the blood of the animals was again examined, and in all of them rings were found. At the same time the blood of ten imported animals of the same breeds which were never injected, but had served as controls, was examined and gave negative results.

Dr Theiler was also able to trace both the rings and the bacillary forms in cattle which at one time or another had been injected with blood from various other animals, and to observe that these forms multiply in an immune animal when it is placed under adverse circumstances as, for instance, when it is attacked by some other disease.

In the opinion of the author, the foregoing observations indicate that the presence of ring-shaped and rod-shaped endoglobular parasites is due to a previous infection of ordinary red-water. Seeing that cattle suffering from tropical piroplasmosis only recover to the extent of 5 per cent., the thought that East Coast fever was complicating the foregoing experiments must be excluded. In addition to this, the case of the first-mentioned ox (No. 6) clearly demonstrated that when small rings and bacillary forms have once been present in rather large numbers (10 per cent.), such an animal, when exposed to tick infection, will succumb, since it has not acquired any immunity against tropical piroplasmosis; neither does the presence of rings in the blood of animals indicate that immunity must be expected against East Coast fever.

The ring-shaped and bacillary-shaped piroplasmata have been overlooked hitherto in connection with ordinary redwater. That may be attributed to the fact that the examination of blood of animals suffering from ordinary redwater has not been continued long enough, but also to the fact that the parasites stained with ordinary aniline dyes are not easily recognised as such, and, finally, because in the majority of cases they are so rare that they are easily overlooked. The typical stains for protozoa, such as Romanowsky's modification, are necessary in order to recognise the chromatic nucleus, which removes all doubt as to the true nature of the endoglobular parasite.

The ring-shaped and bacillary-shaped piroplasmata in immune cattle were considered by Professor Koch to be identical with what Dr Theiler has termed the piroplasma parvum of tropical piroplasmosis. Koch has also observed that the inoculation of blood taken from a sick animal suffering from tropical piroplasmosis does not produce the disease in the new animal. When, however, the inoculation is repeated, the appearance of the usual small parasites in the blood is noticed in some of the animals; and he concludes that, under certain conditions, the organisms of East Coast fever may multiply and undergo reproduction in the blood of the injected animal, notwithstanding that there is no reaction. Furthermore, he states: "Other experiments have also shown that inoculations with the blood of recovered animals, which only contains an inconsiderable number of single parasites, will induce similar modified attacks of African Coast fever, and while these experiments have not been numerous, they tend to indicate that recovered animals are even more suitable for inoculation purposes than those which are actually sick."

At different intervals the author injected animals immune against South African redwater with virulent blood of tropical piroplasmosis, and also with blood of animals immune against this disease, in quantities up to 2000 cc. at a time. After intervals of varying duration these animals were exposed to natural infection, when they all, with only one exception, contracted the disease and died. The injection of blood from an animal suffering from tropical piroplasmosis into another one may, indeed, cause the rise of the temperature, and very often after the reaction basic points and basophile red corpuscles are noticed, together with the appearance of rings and bacillary piroplasms. Of five Texas calves which were injected on 24th November 1903 with quantities of 10, 50, 100, 500, and 1000 cc. virulent blood (East Coast fever blood), all showed reaction due to the injection. The inoculated blood was derived from another Texas calf whose blood was swarming with the piroplasma parvum. The animals were later examined for rings, when it was found that the bacillary and ring-shaped piroplasmata were present; and in the animals alive to-day they are still found. The calf which was injected with 1000 cc. virulent blood was infected on 8th February 1904 with only two pathogenic ticks (rhipicephalus appendiculatus). It contracted the tropical piroplasmosis, and after an illness of thirteen days died from the disease. In this case it is uncertain whether the rings were not already present before the injection, no previous examination having been made of the blood. The appearance may be attributed to the injection of virulent blood; but, whatever the cause of these rings, their presence does not indicate the immunity of the animal against piroplasma parvum.

When one analyses the experiments made by injection with tropical piroplasmosis blood or blood of the immune animal, it becomes evident that with the piroplasma parvum we also inject the organisms of ordinary redwater in its immune form. All animals which have hitherto recovered from tropical piroplasmosis are immune against redwater. East Coast fever has only so far been observed in redwater countries. The increase of rings and bacillary forms of piroplasma bigeminum may be a result of such injections, even when an animal is already immune against redwater.

Conclusions.—(1) The injection of blood into a susceptible animal, taken from an ox immune against ordinary redwater or Texas fever, usually gives rise to a primary and a secondary reaction, during which period the typical pear-shaped piroplasma bigeminum makes its appearance.

(2) Either with the second reaction or shortly afterwards endoglobular parasites appear in the shape of rings and rods, which resemble the piroplasma parvum of East Coast fever.

(3) These rings and rods are seen in the majority of all cattle living in a redwater-infected area and which at one time or another have been injected with blood of cattle immune against redwater.

(4) The presence of the rings and rods in the blood of an ox proves immunity against redwater.

(5) When rings are observed in cattle injected with blood of cattle suffering from or immune against East Coast fever, then they are due to the simultaneous injection of immune blood of ordinary redwater. At the present time East Coast fever exists only in redwater-infected areas, where the cattle are immune against redwater.

(6) The presence of rings does not indicate that an animal is immune against East Coast fever.

(7) The rings and rods may, under certain conditions, multiply and increase in numbers.

(8) The rings and rods represent a phase in the life-history of piroplasma bigeminum in the immune ox.

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IMPLANTATION OF TISSUE AND ITS RELATION TO CANCER.[1]

(Continued from p. 56.)

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III. THEORIES AS TO THE BIOLOGICAL ORIGIN OF CANCER.

Opposed to those who believe that the epithelial proliferation in cancer is due to the action of a parasite are many men who believe that it is due to the development of inherent or acquired peculiarities of the cells themselves, and that the proliferation of the tumour cells can be explained on biological grounds. Various hypotheses have been advanced to explain the unlimited growth of the epithelium in cancer or the tissues of other malignant tumours, with the formation of metastases. The hypotheses which have received widest acceptance are those of Thiersch, of Cohnheim, and of Ribbert, to which may now be added the hypothesis supported by Bashford and Murray. Each of these hypothesis is supported by a greater or less number of clinical facts, and Cohnheim's and Ribbert's have been tested by a considerable amount of experimental work.

Thiersch[2] observed that some benign tumours, *e.g.*, warts, were composed of epithelial cells and a connective-tissue stroma, and yet that the same elements entered into the structure of malignant cancers, in which infiltration of surrounding tissues by epithelium was the marked feature. He therefore decided that the mere presence of epithelial cells alone could not be accountable for the infiltrating peculiarity of cancers. He therefore assumed that a balance of the tissues in the body was maintained by a continual exercise of mutual pressure of one tissue upon another, and that the maintenance of this balance depended upon the perfection of the nutrition of each of the adjacent tissues. Hence if any given tissue had its nutrition interfered with it became unable to maintain its relative pressure, and so might be overwhelmed by adjacent tissues which retained their nutrition, *e.g.*, in old people for some inherent reason the connective-tissue failed to maintain its nutrition, hence the resistance of the tissue was lessened and an ingrowth of adjacent epithelial cells occurred. Hence the liability of old people to cancer. The hypothesis of Thiersch's seems to me hardly entitled to be designated as a "theory," but to be merely a restatement in general terms of a well-known clinical fact, *i.e.*, the infiltration of connective-tissue by epithelial cells in cancer. Moreover, even if the hypothesis were a correct one, unless the real cause of the loss of nutrition could be determined, not the slightest light would be thrown on the cause of the disease. Moreover, beyond the general statement, the hypothesis fits but a small number of the known clinical facts.

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[1] Reprinted from the "Journal of Medical Research," Jan. 1905.

[2] Thiersch: Der Epithelialkrebs. Leipzig, 1865.

Cohnheim's[1] hypothesis, however, has a much more impressive support by clinical and experimental facts. Up to Cohnheim's time great stress had been assigned to trauma as a cause of cancer. Cohnheim believed that no importance could be assigned to traumatism as a cause of the disease. It is perfectly well known by histological and experimental study what the reaction of tissue to traumatism is, *i.e.*, it leads to replacement of the injured tissue by granulation tissue, while the surface is covered by newly-formed epithelium. Now to assume that trauma may cause cancer is to assume that the organism suddenly entirely changes the laws under which it ordinarily acts, and that, too, in an individual who previously has reacted to trauma in accordance with the regular laws many times. Moreover, many cancers develop in places in which no known trauma has occurred. A study of the cases of cancer in various clinics has shown that in a very considerable number of cancers no traumatism could be assigned. Although in some cases cancer is recognised soon after a traumatism, the relative frequency of such cases can be known only by knowing the entire number of traumatisms which occurred during the same period and the number of times that cancer appeared in that entire number. This number obviously would be exceedingly small. Hence the cases of cancer in connection with trauma either could perfectly well be explained as coincidences, or else some accessory cause other than trauma must be assumed to cause the sudden reversal of normal laws in reaction to injury.

Hence Cohnheim, declining to accept the theory of traumatic origin of cancer, assumed that in each of the primary embryonic layers more cells were produced than were necessary for the development of the adult tissues, and that some of these surplus embryonic cells did not develop into the normal tissues of the body, but persisted as "rests" which have the "potentiality of growth" characteristic of fœtal cells, although they presented no gross or histological peculiarities which would permit of their recognition.

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[1] Cohnheim: "Allgem. Pathologie," zweite Anlage, Berlin, 1882, p. 732.

These supposititious "rests" once produced have an ultimate fate dependent upon various accessory conditions which may or may not act as a stimulus to their further development. It would be conceivable that an individual in whom these "rests" developed might grow to adult life and die without any accessory cause ever stimulating them to unlimited growth. Or the proliferation of these "rests" might begin at a very early stage of fœtal life, and in this way the occurrence of congenital tumours could be explained. Or the accessory causes might become active during any period of life, early childhood, or young adult life, or the "rests" might remain quiescent during many years and be excited to growth only during later life, after, *e.g.*, according to Thiersch's idea, the normal resistance of some of the tissues had become lessened. Thus could be explained the occurrence of exostoses at the time of greatest bone activity, or of ovarian cysts at puberty, or of breast tumour during pregnancy, or the occurrence of cancer in late adult life, all well-known clinical facts. The number of examples could be multiplied. Moreover, the constant occurrence of certain tumours in certain regions, *e.g.*, cancers of the lip or of the rectum, might be explained by such hypothetical "rests," as the production of such rests would be more likely to occur at such sites of active embryonic cell proliferation.

This "rest" theory also would explain the occurrence of heterologous tumours, *e.g.*, chondromata in bone, which differ widely from the normal tissue in which they occur. All of the connective-tissues are derived from the mesenchyma. Mesenchymal rests might be formed at any period of the development of these tissues. If they were stimulated to growth and continued to form cells of the period at which they were formed they might, *e.g.*, form tumours of cartilage in the midst of bone. In the same way certain sarcomata, *e.g.*, the myxosarcomata, resemble histologically certain stages in the development of normal connective-tissue. By Cohnheim's hypothesis such tumours could arise from a proliferation of the fœtal rests with retarded development.

The attractive part of Cohnheim's hypothesis is the fact that the same explanation serves to account for the occurrence of benign and malignant tumours. It also is in accord with a large number of known clinical facts. But although this hypothesis might be acceptable as explaining the origin of tumours, yet it in no way accounts for the cause of the sudden activity of these rests, which clinically may start up at any stage of intra- or extra-uterine life. To meet this objection, Cohnheim suggested the possibility that an increase of the local blood supply, combined with a supposed diminished resistance of the adjacent normal tissues, due to a physiological limit of growth, might determine the sudden activity of these supposititious rests.

In support of Cohnheim's hypothesis is a certain amount of experimental evidence. Leopold[1] implanted both epithelial tissue and cartilage from fœtal and adult animals. In many cases the implanted tissues disappeared or became encapsulated; in some cases they grew. Tissue from either young or adult extrauterine animals showed relatively little tendency to grow. Tissue from fœtal animals showed much greater power of growth, and, roughly speaking, the younger the fœtus the greater this tendency, and some fœtal tissues by growth produced a nodule a thousand times greater than that of the original mass, and formed what Leopold considered a true "tumour." Fœtal cartilage thus implanted showed great proliferation of cartilage cells, and also developed in some cases into true bone. In no case did metastases appear, nor was there any evidence of infiltration of adjacent tissues. The transplanted tissues in some cases preserved the vitality and power of growth for very considerable periods. Zahn[1] implanted rib cartilage from adult animals, and found that the cartilage became encapsulated and did not grow. Implantations of fœtal cartilage, however, showed marked growth of the cartilage cells. Cohnheim and Maas[2] put a piece of adult periosteum in the jugular vein of fowls, and found that these artificial emboli in the early stages showed active growth with formation of cartilage and bone, but after a few weeks all of the nodules were resolved and disappeared. Lack[3] opened the peritoneum of rabbits and scraped the ovaries, thus setting free ovarian epithelium. In one animal at the end of fourteen months there was found an undoubted columnar-celled infiltrating cancer of the uterus with a nodule infiltrating the diaphragm, and with metastases. On account of the great rarity of cancer in rabbits this case is particularly striking, but neither Lack nor others so far have been able to repeat the experiment successfully. Frænkel[4] implanted the placenta of rabbits into the same animal or into others, but got no results. He also implanted a rabbit with the sexual organs of her own fœtus, and also repeated Lack's experiment, but in no case did any tumour result. These experiments show the possibility of transplanting certain fœtal or adult tissues, entirely removed from their original environment, into other parts of the same animal, or into other animals, and that in a certain proportion of the cases the transplanted tissue can maintain its integrity, grow, and reproduce the original type of tissue, in some cases for an indefinite period. In but one case was any one of these experiments followed by a malignant infiltrating tumour with unlimited power of growth, and this case, in consideration of the impossibility of repetition, must be considered as only a coincidence.

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[1] Leopold: Experimentelle Untersuchungen über die Atiologie der Geschwülste. "Virch. Arch.," LXXXV., p. 283, 1881.

As further clinical evidence in favour of Cohnheim's hypothesis should be mentioned the occurrence of malignant tumours of the kidney composed of cells which reproduce in type the cells of the adrenal. The occurrence of such tumours suggests the probability that the tumours arise from an inclusion in the kidney of masses of adrenal cells during the embryonic period. Also the occurrence of syncitial tumours of the uterus, "deciduomata maligna," which appear in the uterus during or near pregnancy, are composed of cells which arise from the epithelial membranes of the ovum, which enter and infiltrate the uterus, remain for a time purely local, but ultimately become disseminated by the blood and form metastases in distant organs, is evidence infavour of the theory.

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[1] Zahn : Sort des tissus implantés dans l'organisme. "Congrès period. internal de Genèvre, C.R.," 1878, p. 658.

[2] Cohnheim and Maas : Zur Theorie der Geschwulstmetastasen. "Virch. Arch.," LXX.

[3] Lack : A preliminary note on the experimental production of cancer. "Jour. Path. and Bact." VI., p. 154.

[4] Frænkel : Ueber Versuche, durch experimentelle Verlagerung von Keim Gewebe Karzinom zu erzeugen. "Centralbl. f. Allgem. Path.," XIV., p. 664, 1903.

Ribbert's theory.—Ribbert,[1] from a histological study of very early cancers believes that the epithelial proliferation is due primarily to a separation of epithelial cells from their attachment to the normal epithelial layers by the action of the connective-tissue. These freed epithelial cells, still retaining their power of growth, get into the clefts between the connective-tissue cells, proliferate, and thus form islands of epithelial cells, which, freed from the normal restraining influence of other tissues have an unlimited power of growth. There is some evidence, clinical and experimental, that normal epithelium, set free by accident or design from its original attachment, may retain its power of growth and produce nodules or cysts of epithelium, but there is no proof that true malignant tumours can be so produced, *e.g.*, repeatedly there has been reported the occurrence of cysts, "traumatic dermoid cysts," lined with epithelium, occurring in human beings, chiefly on the hands or face, subsequent to injury. It has been shown by histological study and by experiment that such cysts are due to accidental separation of surface epithelium, with implantation in the depths of a wound. Such cases have been reported by Wegner[2] Bohm,[3] Le Fort,[4] Garré,[5] Blumberg,[6] Paulet,[7] Reverdin,[8] and Gironde.[9] Similar lesions have been seen in the iris and vitreous of the eye after injuries to the cornea. On the hands or face the nodules appear either as masses of pearly epithelium or as cysts lined with epidermis containing sebaceous material. The walls consist only of epidermis as a rule, and do not contain hair follicles or glands. Schweininger[10] and Kauffmann[11] produced such cysts by implanting small bits of epidermis in the tissues of animals, but found that while at first the rate of growth was very rapid, after a time the rate diminished and the increase in size was very slow, *i.e.*, they produced epidermoid cysts which did not have the essential character of malignant tumours.

On the whole, in regard to the biological theories of the origin of malignant and benign tumours as enunciated by Thiersch, Cohnheim, and Ribbert, it may be said that such hypotheses, or a combination of them, is at present supported by a considerable number of clinical and experimental facts; that certain tumours, dermoid cysts, can be produced experimentally under the conditions of the hypotheses, and that in one doubtful case a truly malignant tumour appeared, and finally that fœtal tissues undoubtedly have a much greater inherent power of growth than do adult tissues.

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[1] Ribbert: Beiträge zur Histogenese des Carcinoms. "Arch. f. Anat. u. Physiol.," CXXXV., p. 433, 1894. Also, "Zur Entstehung der Geschwülste. Dtsch. Med. Wochenschr.," 23rd July 1896.

[2] Wegner: Beiträge zur Lehre von den traumatischen Epithelcysten. "Relf. Centralbl. f. Allgem. Path.," X., p. 581, 1899.

[3] Bohm: Traumatische Epithelcyste und Fremd-körper Riesenzellen in der Haut. "Virch. Arch.," CXLIV., p. 276.

[4] Le Fort: Des Kystes Dermoïdes Traumatiques. "Révue de la Chir." 1894, p. 1013.

[5] Garré: Ueber traumatische Epithelcysten der Finger. "Beiträge zur klin. Chir." XI., 1894.

[6] Blumberg: Ueber sogennante "traumatische Epithelcysten." "Dtsch. Zeitsch. f. Chir.," XXXVIII., p. 605.

[7] Paulet: Des kystes dermoïdes acquis. "Bull. et Mém. de la Soc. de Chir.," Paris, XII., p. 460, 1886.

[8] Reverdin: Des Kystes Epidermiques des doigts. "Rev. Méd. de la Suisse romande," 1887, p. 121.

[9] Gironde: Contributions à l'étude des kystes dermoïdes. "Lyon Medical," 1879.

[10] Schweininger: Beitrag zur Erzeugung Hautgeschwülsten. "Charité Annalen," XI., p. 642.

[11] Kauffmann: Ueber Enkatarraphie von Epithel. "Virch. Arch.," XCVII., p. 236.

Too lately to allow of verification by other men, certain observations have been made which give support to the theory that cancers may be considered to be a parasitic individual engrafted on a normal individual, and that they are produced by the conjugation of cells in a way analogous to conjugation of sexual cells which produce a normal individual. Farmer, Moore, and Walker[1] investigated cancers, and concluded that the reduction in the number of chromosomes in the mitosing cells took place in the same way as in the ripening of the sexual cells of animals and plants, and that the number of these chromosomes were half as numerous as in the somatic cells. They studied not only human cancers, but also cancers in various lower animals. Bashford and Murray[2] confirmed these observations. These observations show a series of changes in the nuclei of malignant tumours throughout the whole extent of their known zoological distribution parallel to those characteristics of the maturation of the sexual elements of the metazoa. If these observations are confirmed, it will be a most important step in elucidating the origin and development of tumours.

My own implantation experiments were undertaken bearing in mind the above presented evidence, except that since published by Bashford and Murray, with the belief that evidence of the biological origin of tumours far outweighed that for a parasitic origin. The experiments were undertaken to see if by any means normal epithelium or other tissue, either fœtal or adult, freed from its original environment, could maintain its power of growth, and lead to the formation of anything which could be considered analogous to a tumour, *i.e.*, tissue with unlimited power of growth. About ninety animals in all were used, but only those animals in which no suppuration occurred are reported.

The transplantations were all performed under strict surgical asepsis, the hair being previously removed by the application of "Sulphur Starch" as a depilatory. The animals, except a few which died of various infections, were killed at various times, usually some weeks or months after the operation, with the idea of giving the transplanted cells as long a time as possible for growth. Only rabbits and guinea-pigs were used. In some groups of experiments absolutely negative results were obtained. In others, chiefly with epithelium of the skin, nodules many times the size of the original implantations were obtained, analogous to dermoid cysts, but in no case was a true tumour of metastasis produced.

The experiments fall into groups in accordance with the tissue used.

1. *Implantation of Testicular Epithelium.*

Five animals. In each case the peritoneal cavity was opened, the testis was drawn into the abdominal cavity, the tunica was split, and the testicular substance was curetted and distributed in the peritoneal cavity. The peritoneum was then closed. The animals were killed at periods varying from sixty to 161 days. In no case was there any evidence of growth of testicular epithelium. The peritoneum in each case was normal except for slight adhesions.

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[1] Farmer, Moore, and Walker: Resemblances exhibited between the cells of malignant growths in man and those of normal reproductive tissue. "Proc. Roy. Soc. Vol.," LXXII.

[2] Bashford and Murray: Loc. cit., supra.

2. *Implantation of Ovarian Epithelium.*

Seven animals reported upon. In three cases the peritoneum was opened, the entire ovary, or a half of it, was removed and immediately imbedded in the subcutaneous tissue of the abdomen of the same animal. In each case, for about two weeks, a nodule, larger than the implanted ovary, probably from exudation, was seen in the abdominal wall. The nodule gradually diminished in size and finally disappeared. One animal was killed at the end of sixty-three days, one after seventy-one days, and the other after 101 days. In all cases the only evidence of the implantation was a small spherical mass of dense fibrous tissue. On histological examination no remnant of ovary was to be found, the mass consisting of dense fibrous tissue. In four cases Lack's experiment was repeated, *i.e.*, the abdomen was opened and the ovary crushed or curetted, and the tissue disseminated as widely as possible in the peritoneal cavity, and the peritoneal cavity was closed. The animals were allowed to live sixty-one, sixty-seven, ninety-three, and 210 days respectively. At autopsy no growth or other change in the peritoneal cavity was found beyond slight fibrous adhesion.

3. *Implantation of Renal Epithelium in the Same Animal.*

Four animals were operated upon. In each case a lumbar incision was made, the kidney was drawn out of the wound, and a small bit of the kidney cortex was excised without opening the pelvis of the kidney, and the tissue was immediately buried in the subcutaneous tissue of the same animal. The animals were killed at the end of three, sixty-seven, sixty-eight, and seventy days respectively. In the animal killed at the end of three days the kidney tissue maintained its contour, but showed marked surrounding infiltration with polynuclear leucocytes. The cells of the renal tissue were largely necrotic. In the other three cases the nodules at first rapidly increased in size, probably from the presence of exudation, and then gradually diminished and showed finally only a small spherical mass of dense fibrous tissue, in which, on histological examination, no renal tissue was to be found.

4. *Implantation of Liver Epithelium in the Same Animal.*

Ten animals were operated upon. In two cases a small piece of liver was removed, the liver wound satured, and the removed liver crushed in a mortar in sterile salt solution at body temperature, and immediately injected into the subcutaneous tissue of the same animal. The animals were killed at the end of 120 and 163 days, and showed nothing. In two cases a similar emulsion was injected into the mesenteric vein of the same animal. The animals were killed at the end of 124 and 163 days, and showed nothing. In two cases in pregnant animals, at about half term, the uterus was opened, the fœtuses removed, and from one, while still attached to the placenta, a small portion of the fœtal liver was removed and buried in the subcutaneous tissue of the mother. The uterus then was closed. The implanted nodules, after the disappearance of the exudate, slowly diminished in size, and finally disappeared, showing no hepatic tissue at autopsy. In four cases a small bit of adult liver was implanted in the subcutaneous tissue of the same animal with no result.

5. *Implantation of Adrenal Tissue into Kidney of Same Animal.*

Six animals were operated upon. The abdomen was opened, the adrenal was removed and implanted either in part or entire into the cortex of the kidney of the same size, and the kidney capsule sewed over it. The animals lived from four to eleven days. In each case the adrenal tissue was necrotic, showed some infiltration with leucocytes, as did adjacent renal tissue, and in the cases which lived longest the beginning of connective tissue encapsulation was seen, but in no case was there any evidence of adrenal proliferation.

6. *Implantation of Adult or Fœtal Epidermis into the Same Animal.*

Eleven animals were operated upon, ten with adult, one with fœtal epidermis. Rabbits were used entirely. A very small bit of epidermis from the ear, about as large as the head of a pin, was removed with fine scissors, and care was taken not to get corium. In most cases no hæmorrhage occurred. This epidermis was immediately pushed into the subcutaneous tissue of the ear on the outer side of the cartilage. In this way the gross changes could be fairly closely observed on account of the transparency of the ear. In the course of a few hours the nodule assumed a more or less spherical form from exudation of hæmorrhage, and increased in size. In the course of ten to fourteen days the nodules became smaller and white. Then came a fairly rapid increase in size, the spherical shape being retained, and then, in two or three weeks, the nodules either increased in size slowly or remained of the same size. The animals were killed in periods varying from seven to 270 days. On section the nodules showed either a firm consistency, or appeared as small cysts containing a whitish or yellowish grumous material. The larger nodules were perhaps twenty times as large as the piece originally introduced, but in all cases there was a marked increase. On microscopic examination it was seen that in every case the epithelium had proliferated and produced a spherical nodule of epithelium or a spherical cyst lined with epithelium. When the nodule was solid the entire mass was composed of epithelium, the inner layers being composed of laminæ of cornified epithelium, while the outer layer of cells was the same as the deeper layer of cells in normal epidermis. Between the inner and outer layers the layers of normal epidermis were reproduced. In the larger nodules, the true cysts, the centre was a more or less spherical cavity lined with epidermis, the outer layer of which was like the deepest layer of the normal skin, while the innermost layer was composed of stratified cornified epithelium. The central cavity was filled with material like that seen in a wen *i.e.* granular material and broken down cornified epithelium, with, in some cases, hairs. In many of the cysts hair follicles and sebaceous glands can be seen in the wall of the cyst, but in no case are such follicles or cysts seen about the entire circumference, showing that epidermis had grown across the surface of the small wound of implantation, thus producing the spherical form, but that the new epidermis did not specialize to form hair follicles or glands, *i.e.*, the epidermis acted like the epidermis of a superficial wound. In all cases the nodule or cyst was surrounded by a fairly distinct layer of dense fibrous tissue. In no case was there produced any isolated nodule of epidermis, nor was there any evidence of infiltration of the surrounding connective tissue, nor was there any extension of epidermis along lymph sinuses to adjacent lymph nodes.

In one case epidermis from a fœtus at about half term was used, and immediately implanted into the subcutaneous tissue of the ear of the mother. In this case the resulting nodule was relatively larger than in those cases where adult epithelium was used, but the histological picture was identical with that produced by the implant of adult epithelium. In many cases proliferation of the epidermis was rapid, as was shown by frequent mitotic figures.

So it will be seen that by these experiments nodules or cysts were reproduced, analogous to the "traumatic epithelial cysts" already mentioned, and also analogous to dermoid cysts, but in no case was a true infiltrating tumour produced. In many cases the "potentiality of growth" was retained by the epithelium to a marked degree, especially so when fœtal epidermis was used.

7. *Implantation of Adult Uterine Epithelium, Pregnant and Non-pregnant, into the Same Animal.*

Ten animals were operated upon. In three cases the animal was pregnant when the transplantation was done. In seven animals the uterus was in normal condition. The abdomen was opened. If the animal was pregnant the uterus was opened and the fœtuses were emptied out, together with the placental attachments. A cross section of the uterus, one-half centimetre long was then removed, and immediately implanted into the abdominal subcutaneous tissue of the same animal. The piece removed was in no case through the placental attachment. The uterus was then closed by suture, as was the abdominal wall and the wound of implantation. The nodule was of considerable size, and its gross changes could be followed. There came in each instance a marked increase in size from exudation. This first increase in size then gradually diminished, and the nodule either finally disappeared or, after two or three weeks, gradually increased again, until in one instance it attained a size ten times as large as that of the original nodule. On gross examination the nodules were generally irregular and rather elastic. On section the nodule was invariably cystic, either showing one cyst cavity, filled with a cloudy yellow fluid, or in some cases showing multiple cysts. On histological examination the appearances were fairly constant within certain limits of variation dependent upon the rate of growth of the transplanted tissue. The cyst cavity in every case was lined with epithelium more or less closely approximating that of the normal uterine mucous membrane. In all cases the number of mitoses was small. In the smaller tumours the epithelium was almost columnar, and cilia were distinct. In the larger cysts the epithelium was more or less flattened, and cilia were indistinct. The walls of the cysts were in all cases irregular in outline, varying from coarse polypoid projections to complicated projections, giving a picture like that of a multilocular cyst. In some cases but one cyst cavity was seen, corresponding to the lumen of the transplanted horn of the uterus. In other cases several cavities of large size were seen, each as large as the cavity of the transplanted uterus. In one case where the transplanted tissue was taken from a pregnant uterus there appeared on one side of the resulting cyst very marked hyperplastic endometritis of the mucus forming a hemispherical nodule on one side of the cyst, the rest of the cyst being lined with a single layer of epithelium.

Directly outside the mucous membrane in most cases was a layer of loose connective tissue, with in most cases considerable infiltration with lymphoid cells (submucous connective tissue). Outside of this layer in many cases was a layer of smooth muscle fibre, corresponding to the normal smooth muscle of the uterus. In some cases this muscle was almost perfectly preserved, and showed practically no formation of scar tissue. In other cases this smooth muscle layer was less than normal, and in it there were extensive areas of dense connective or scar tissue. In other cases this muscle layer was extremely thin, due in many cases apparently to the great dilatation of the cavity of the transplanted uterus. About some of the larger cysts in some of the nodules no smooth muscle at all was to be seen. This probably is due to the fact that the epithelium of the mucous membrane acted as does surface epithelium when transplanted, *i.e.*, it grows over the surface of the wound of implantation, and in that way forms a cavity lined with epithelium. Since the growth of epithelium is irregular, the nodule on section shows adjacent cavities which at first give the appearance of a multilocular cyst.

Outside of the muscular layer in all cases comes a layer of fibrous tissue, in some cases wide and clearly marked and composed of dense scar tissue and forming practically a capsule; in other cases, indistinct, and distinguished with difficulty, if at all, from the normal loose connective tissue, and forming no encapsulation at all. In no case was there any evidence of infiltration of adjacent tissues by the uterine epithelium, nor any evidence of metastases by extension along lymphatics to adjacent lymph nodes. The transplanted nodules in all cases maintained their "potentiality of growth," as shown by occasional mitoses and gradual increase in size for considerable periods of time, but nothing comparable to a malignant tumour was produced.

8. *Implantation of Placental Tissue into the Same (Maternal) Animal.*

This series of experiments was undertaken to see if anything analogous to the deciduoma malignum could be produced. Besides the attempts to transplant placental tissues direct, in all of the preceding class of uterine implantations, whenever the animal was pregnant the uterus was manipulated and massaged to see if it were possible to produce any absorption of placental tissue by the uterine blood vessels which would lead to placental metastasis. In no case did any such metastasis occur. In six animals direct implantations of placental tissue into the subcutaneous tissue of the same animal were undertaken. In three of these cases the uterus was opened late during the animals' pregnancy, when the fœtuses were almost at term, and small bits of placenta were removed and immediately implanted into the mothers' subcutaneous tissue. In all three cases the result was the same. The nodule persisted for a time, and showed the usual early increase in size from exudation, with a later gradual diminution and final disappearance of the nodule. In three cases the uterus was opened very early in the pregnancy before distinct development of fœtal outline, and the entire placenta was implanted in the mothers' subcutaneous tissue. In each case the nodule ultimately disappeared.

9. *Implantation of an Entire Fœtus into the Same (Maternal) Animal.*

Two animals were operated upon. In both cases the uterus was opened early in the pregnancy, and the fœtus, about one-half centimetre long, was removed from its placental attachment, and with the yolk sac adherent immediately implanted in the mothers' subcutaneous tissue. In one case the nodule ultimately disappeared. In the other case the fœtal tissues retained their power of growth and the nodule gradually increased in size and, at the end of ninety days produced a nodular mass at least ten times the bulk of the original fœtus. On section this nodule showed several small irregular cysts containing clear glairy fluid, and the cysts appeared not to connect with each other. On histological examination a very complicated picture was found, analogous to that of a complicated teratoma. No definite fœtus could be distinguished, but there was a remarkable mixture of cysts lined with epithelium, cysts due to necrosis, and areas of bone and cartilage. The entire mass of transplanted and growing tissue was surrounded by a layer of dense fibrous tissue. In places this fibrous tissue layer was thin and hardly to be distinguished from the normal subcutaneous connective tissue. In other places it was thick and dense. One of the larger cavities of the mass was due to necrosis of a considerable portion of the nodule. The walls of this cavity were composed of granulation tissue. The contents was necrotic tissue, the character of which could not be determined, with serum and many polynuclear leucocytes. Other cysts were lined with epithelium and glands like the lining of the intestinal tract, outside of which was a layer of lymphoid tissue, outside of which again was a layer of smooth muscle fibre. Of these fibres only one layer could be distinguished. Many of the epithelial cells lining the cysts were beaker cells, and in all cases the glands of the inner walls of the cyst were like the glands of the normal intestine. In places were also large islands of hyaline cartilage, scattered irregularly about, and in other places were small areas of bone with trabeculæ and marrow spaces filled with marrow. The remarkable feature about this nodule is the fact that in a normal fœtus the size of this implanted one the intestinal tract is represented by a tube lined with a single layer of columnar epithelium with no differentiation of glands or muscle, and, moreover, no differentiation of the mesenchymal tissues into bone has taken place, while in the nodule produced by implantation tissues closely resembling adult intestine and adult bone and cartilage have been produced. The nodule was slowly increasing in size at the time the animal was killed in order to complete this report.

10. *Implantation of Fœtal Cartilage into the Same (Maternal) Animal.*

But one animal was operated upon. A pregnant rabbit, with fœtus at about half term, was operated upon by opening the uterus, removing a fœtus, and immediately transplanting a small bit of the cartilage of the femur, in which no ossification had taken place, into the subcutaneous tissue of the mother. As usual the nodule increased, grew smaller, and then gradually increased in size until a nodule more than ten times the size of the original nodule was produced. Histological examination showed an oval nodule, surrounded by a distinct capsule of dense fibrous tissue. Inside this capsule was a nodule partly bony, partly cartilaginous. The periphery was composed of a layer of fibro-cartilage. Inside of this layer was a layer of hyaline cartilage with very large cartilage cells, occurring singly and not in large groups. Inside of this hyaline cartilage, and occupying about one-third of the entire nodule, was an area of true bone, with trabeculæ in which were cells in transition stage between cartilage cells and bone corpuscles. The marrow was very vascular. The transition from bone to cartilage was much like the series of changes which take place at the epiphyseal line in the ossification of the long bones.

CONCLUSIONS.

1. Certain types of epithelium (epidermis), both adult and fœtal, can be experimentally removed from their normal position and implanted into another part of the same animal, and under those circumstances can maintain their "potentiality of growth," retain their own peculiar character, and produce nodules analogous to dermoid cysts or more complicated teratomata.
2. In no case has any epithelium of a highly differentiated function been seen to maintain its power of growth or to proliferate.
3. The "potentiality of growth" is greater in the case of fœtal than it is in that of adult tissues.
4. In no case has any infiltration of surrounding tissue by the transplanted epithelium been seen, nor any tendency to epithelial metastases.
5. Certain fœtal connective tissues (cartilage) can be transplanted in the same way as epithelial tissues, and retain their "potentiality of growth."
6. Transplanted fœtal tissues do not represent the stage of development at which they are transplanted, but tend to reproduce the ultimate stage of their normal development.

SOME OBSERVATIONS ON TROPICAL BILIARY FEVER.

By MAJOR F. EASSIE, D.S.O., A.V.D.

THE article by Dr Edington in the March number of this *Journal* suggests a point in connection with tropical biliary fever which it would appear has not as yet been accorded sufficient prominence. I refer to its existence not only as a primary disease but, under certain conditions, in the form of recurrences.

Perhaps a more definite instance of primary infection could hardly be given than an outbreak that occurred at Port Elizabeth in January 1900. During that month and the month that preceded it large shiploads of mules were landed from Italy and the United States. Taken generally, they were in excellent condition. On arrival each shipload was sent to a farm on the coast, some distance from the town, and by the end of January there were nearly 3000 mules collected there. They were turned out in large camps and left out day and night without shade or shelter. The first animals had not been on the farm more than a week or two when it became evident that the place was infested by ticks. These were of several varieties, but the blue tick (R. decoloratus) greatly predominated. Later on the bulk of the mules became covered with them.

About a month from the date of the first occupation of the farm cases of biliary fever began to occur, and as time progressed it became evident that it was making considerable headway. Provision was accordingly made for the care of the large number of sick, and representations were made stating the necessity for the removal of the remainder. It was, however, not until the end of February that their removal could be effected, by which time quite one-third of them had been attacked. Meanwhile, fortunately, some large sheds were secured in which to place the sick.

It may be said at once that the exact nature of the disease was not understood. At that time the researches of Dr Theiler had not been disclosed. It was, however, evident at least that the disease was malarial. The prominent symptoms were: rise of temperature rarely exceeding 103°, brownness of the visible mucous membranes, hæmolysis evidenced by hæmoglobinuria, and angular blotches on the conjunctivæ—intensely purple. What, however, particularly characterised this outbreak was the degree of prostration that succeeded the first few days of fever. In this phase of the disease the mules were quite unable to rise, and we had frequently as many as thirty at one time lying utterly helpless on thick beds of rushes. These were turned over from one side to the other, as they lay, continuously; receiving, the while, ammonium chloride and belladonna, and taking the dose with characteristic absence of any power of resistance.[1] From this stage they recovered in a few days sufficiently to be able to stand in slings. These we were fortunately able to requisition from horse transports in the Bay. After a few days standing in the support of slings, they were, as a rule, able to stand alone. They were then placed loose in the shade of another large shed under which thick bedding was laid.

Amongst mules that had thus far recovered there were during this outbreak quite twenty cases of luxation of the patella, which in five cases was bilateral. This occurred apparently as the result of atonic relaxation of muscle, and it was in itself a criterion of the degree of prostration.

When orders were finally received at the end of February, all the mules which were able to travel were sent in to the show-yard at Port Elizabeth, where they were tied up in sheds. Amongst the many it is only reasonable to suppose that there must have been a large number which were the subjects of infection and in which the disease had not as yet been declared. This is the more likely as there had been no abatement in the number of cases admitted to treatment up to the time of their removal to Port Elizabeth.

It is a point of some importance to note that the disease vanished with the removal of the mules to the show yard, and that, although the bulk of them had become poor whilst on the farm, they quickly regained condition, and were sent up country before the middle of March.

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[1] The treatment recommended in a pamphlet by Mr Hutcheon.

The heat at Port Elizabeth was intense, and from the direct rays of the sun these mules whilst at the farm had no remission. I am not prepared to say to what extent this may react on horses or mules in health. I am convinced, however, that exposure, without the option of shade, to a tropical sun over a sufficient period is a determining factor both in the appearance and intensity of primary equine piroplasmosis and to even a greater degree in its recurrence. Of the last assertion I hope to offer proof later on.

Before leaving the subject of this outbreak I should like to draw attention to the almost complete absence of mortality. Whether this was due to Mr Hutcheon's prescription or to the fact of their being placed under favourable conditions may be open to question.

My own experience of obviously primary cases of equine piroplasmosis is limited, I feel sure, to the outbreak I have described, and to a few cases, perhaps, that I saw in 1904 at the Remount Depot at Mooi River. The rest of thousands of cases which I saw were, I think, undeniably exacerbations of latent piroplasmosis, determined more than anything else by exposure to the pitiless rays of a tropical sun, daily and without remission; and aided, it is more than probable, by such accidental conditions as, for instance, lack of sufficient water, and debility, the result of over exertion or the want of food.

Before proceeding further it will be as well to summarise the facts of the Port Elizabeth outbreak. Here were large numbers of mules, newly landed, presumably never before the subjects of piroplasmosis, placed in the notoriously malarial coast country of South Africa, on a farm infected by ticks to a very marked degree, the most abundant being the blue tick which it has been inferred, rather than proved, is the necessary carrier and transmitter of the pirosoma; the mules thus exposed, in the hottest weather, without option of any shade, contract what is obviously piroplasmosis in the shortest period possible by natural infection. After the declaration of the disease in quite one-third of the total number of 3000, the remainder are removed from the veldt to covered sheds, and, though many more must logically be supposed to have become infected, there is then a sudden cessation of all clinical evidences. Lastly, the inference drawn from this fact that exposure to tropical sun must have a large influence in the determination of the state of piroplasmosis even in primary infection.

I should like now to draw a picture of what I believe to be the state of exacerbation of occult malaria determined by exposure, of thin animals more particularly, continuously to intense sun heat. One saw hundreds of such cases amongst poor-conditioned horses and mules during the war. They were perhaps loose in camps, often unable to crawl to water, or they were tied up in lines—often in such numbers that their chance of being watered with regularity was exceedingly remote. The skin is parched and hidebound; the temperature often 105°, or higher; the visible mucous membranes are yellow rather than brown; the urine is scanty and high-coloured; and the conjunctivæ are studded with purple angular blotches. What I should like to emphasise is that even in extreme cases these recover if placed under cover and given sufficient water, whereas if left exposed their fate is inevitable. I have had the opportunity of proving this in hundreds of cases.

The question arises: Is this condition a recurrence of occult piroplasmosis? or is it the result of mere exposure of poor-conditioned animals to a tropical sun, aided possibly by lack of water? It is idle now to lament that at the time want of knowledge of the nature of piroplasmosis, and of opportunity for microscopical enquiry, prevented an exact determination of this question. The fact remains that the clinical evidences were so far dependent on exposure to intense heat that they disappeared when relief from it was afforded. Conversely, I have been able to show on more than one occasion that exposure of the poor-conditioned animals returned to me from columns was followed as a rule by appearance of the conditions described. Further than this, the *post-mortem* appearances in such cases suggested malaria in the yellow discoloration of all white tissues; the enlarged spleen; bile-congested condition of the liver, with increased definition of the lobules; enlarged lymphatics surrounded by hæmorrhages; collapsed condition of intestines, with atrophy of their muscular coat; parboiled appearance of the heart muscle; paleness of the muscles generally. Admitted that cases such as the foregoing are relapses into the state of piroplasmosis, they offer the following clinical differences from cases of primary infection:—

(1) The attack does not suggest an acute disease, but rather a progressive debility.

(2) The temperature is often higher—determined undoubtedly by exposure to the sun.

(3) There is no marked degree of prostration.

(4) The conjunctivæ, although covered with purple blotches, are yellow rather than brown.

(5) The temperature drops to normal immediately they are placed in the shade.

With reference to the recurrence of tropical biliary fever, it may well be asked if, taking into condition its malarial nature, anything else could possibly be expected. There is the close analogy of redwater in cattle, the pirosoma of which is carried by the blue tick—which may also be the intermediary in the case of equine piroplasmosis. The pathological picture is identical in both diseases, and etiologically they are hardly separable. Yet redwater recurs under influences that are debilitating when its existence is not even suspected. There is thus the mixed infection of rinderpest and redwater and of redwater and East Coast fever, which led Dr. Koch to confound them in the first instance.

It would appear that Dr. Edington's recently published conclusions on his experiments at Grahamstown suggest rather the recurrence of latent equine piroplasmosis than the inference that he appears to draw from them that there is any other association between this disease and horse sickness. In this connection one is brought to the consideration of the existence of equine piroplasmosis outside of the sub-continent of South Africa. And one may say in passing that it seems hardly reasonable to associate it, as he appears to do, inseparably, with horse sickness, in the light of the knowledge that it certainly exists in India where horse sickness is unknown. There is reason also to believe that Theiler's discoveries in connection with the origin of biliary fever in South Africa will lead ultimately to the recognition of this disease in other tropical and semi-tropical countries. During over three years' service in Burmah I had a large experience of a disease, of the nature of which I was at the time ignorant, both in indigenous ponies and in mules imported from the Punjab and from western China. Looking back on it now, I can come to no other conclusion but that it was the form of equine piroplasmosis which I have described as recurrent. At that time the losses from surra were enormous. When I took over charge of the Myngyan district there had been a history practically of the total loss of a transport of 3000 mules every year on their return from rationing the permanent military ports beyond the Chin Hills border. They acquired the trypanosoma infection in the terai of the hills, and died after the long incubation period on their return to the bases on the Irawady. At anyrate, it was not until their return that they were noticeably infected. It is perhaps not irrevelant to mention that in succeeding years infection was almost completely avoided by the simple expedient of keeping away from certain areas coloured on a map, defined, it may be said, by observation of the fact that in these localities the villagers kept no other animals but Watu buffaloes. These, although they are infested by the trypanosoma, are immune to consequences resulting from their invasion.

Besides the large number of mules and ponies used annually for rationing the hill posts and for periodical punitive expeditions, there were also in the district a number of mules kept for local transport at numerous posts in the plains. I was frequently employed in dealing with supposed outbreaks of surra amongst these. In such instances I found them invariably picketed in the sun in intense heat. The resemblance to cases of surra was so complete that one would have pronounced them to be the subjects of surra, but for the absence of the easily detected trypanosoma. The symptoms, in short, were those precisely that are common to malarial diseases in all species of animals, to the various known varieties of trypanosomiasis, and to plasmodial diseases in man. One knew that the disease was not surra, and, what was of equal importance practically, that the condition would disappear at once by merely affording them shelter from the sun.

I do not remember to have seen either in India or in Burmah in the course of a rather varied experience any cases that resembled the primary cases at Port Elizabeth; but I feel quite sure that those I have described above, as well as many that I saw in India (particularly in the Chitral Campaign), were cases of recurrent piroplasmosis.

I have urged this point because I consider it one of great practical importance. I think it accounts in a great degree for an otherwise unaccountable amount of the debility which characterises military operations in countries wherein, in the light of Dr. Theiler's discovery, we know, or can infer, that equine piroplasmosis is indigenous. Even in the case of animals born in the country and immune, perhaps, to the disease in its acutest form, there is always the liability to its recurrence, just as there is in the case of redwater in cattle. Unfortunately, exposure to intense sun heat, which appears more than anything else to determine its recurrence, is, in a vast country without trees and under conditions of war, often quite impossible to avoid.

From what I saw of Australian horses in South Africa, I believe that the excessive amount of debility amongst them, which was notorious, is probably explainable on the assumption that they were already the subjects of latent piroplasmosis, and that, landing in poor condition, as a large percentage of them always do, they were more likely to be influenced by a hot sun in a treeless country. I remember to have seen a large number of recently arrived Waler horses at Bloemfontein imported for the South African Constabulary, which showed the symptoms described as peculiar to the recurrent form, and which, on my recommendation, were given shelter and forthwith recovered. I remember also at Jalala, in the Chitral Campaign, two squadrons of Waler horses of a native cavalry regiment, nearly every one of which showed the same symptoms during a spell of terrific heat that was experienced there. These also recovered from the moment they were placed under hastily run up mat shelters.

In this connection I look forward to an echo in Australia of the work done by Dr. Theiler in South Africa, just as this has already occurred in India.[1] It would seem the more reasonable to expect this since the closely allied disease redwater is known to be indigenous there, propagated by the probable carrier of the equine pirosoma. If this surmise should subsequently be proved to be correct, it will explain what has been a surprise to many, that Australian horses should suffer so unaccountably when imported into a country like South Africa, differing so little from their own in latitude.

To summarise the foregoing. It is submitted:—

(*a*) That equine piroplasmosis, like redwater in cattle, once acquired, remains latent, and is subject to recurrence.

(*b*) That this is determined by continual exposure to a tropical sun, particularly in the case of animals tied up, the liability being greater if they are already debilitated, and if they do not get a sufficiency of water.

(*c*) That, as in the case of redwater, it probably recurs as the result of mixed infections.

(*d*) That, in the form determined by exposure to intense sun heat, it differs clinically from the acute disease, resembling more the progressive anæmia of surra; and that the temperature, which is usually higher than in the acute disease, falls when shade is afforded, and recovery even in advanced cases is thus determined.

(*e*) Finally, that equine piroplasmosis is a disease of much wider geographical distribution than is at present realised, and that its resulting unsuspected latency is in all probability responsible for much of the debility which is found to be inseparable from campaigns in tropical countries.

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[1] Article by Lieut. Pallin, "The Veterinary Journal," January, 1905.

EQUINE OSTEOPOROSIS.

By W. Robertson, M.R.C.V.S., Department of
Agriculture, Cape Town.

History and Geographical Distribution.

Equine osteoporosis may be defined as a disease affecting the Equidæ, associated with change in the osseous structure, causing distortion of the bones of the head and frequently lameness, and which may under certain conditions become enzootic.

It was first recognised by Varnell, but current European veterinary literature contains very scanty references to osteoporosis, from which fact we may infer that the disease is rare in these countries, or that it has been classed with some other affections of the osseous structures.

Cases are reported from England, Scotland, Germany, France, Russia, and most countries of Europe; it is said to exist in New Zealand as "Big Head," and in many parts of America. On Long Island, New Jersey, and on the rich soils in the Mississippi Valley and Atlantic and Gulf Coasts, osteoporosis often prevails enzootically, and has its recrudesence like Rachitis (Law, Vet. Med.).

W. L. Williams noticed cases on a farm in central Illinois, and Marshall records cases in Philadelphia and Pennsylvania; Berus (in Brooklyn) reports on 200 cases, and states that hundreds of horses die yearly in Brooklyn of osteoporosis, the same being true of New York City and Philadelphia. In Cincinatti Meyer has found a few cases.

Harold Elliot, M.R.C.V.S., Hilo, Hawaii, writes in the Journal of Comparative Pathology and Therapeutics (Volume XII, p. 300) regarding the nature, cause, and prevalence of the disease amongst the horses of that Island.

In this Colony, particularly the Cape Peninsula, and in certain parts of the Transvaal, the disease is very common, and on the increase in a most marked degree. It is very prevalent in Johannesburg, Port Elizabeth, and the Cape Town district, and particularly common along the Liesbeek River and the low-lying coast line of Mouille Point. The disease first came under the notice of the Chief Veterinary Surgeon of this Colony during the years 1885-89, when he observed it in a well managed racing stable at Port Elizabeth.

"The first three cases, by a curious coincidence, occurred in a colt and two fillies the progeny of the same sire, which led me at first to think that there might be some family predisposition, but subsequently the disease appeared in other horses which were introduced into that stable, and which came from different breeders and had no family relationship" (Hutcheon).

Since that date isolated cases of the disease have been observed. In 1898 a serious outbreak occurred at the Military Camp, Wynberg, C.C., amongst a number of horses which were recently landed from the Argentine. Captain Lane, A.V.D., who was in veterinary charge, supplied the history, which was briefly as follows:—

These horses on arrival here had to undergo a complete change of diet, having been reared on grass and lucerne and fed on lucerne during the voyage. On their arrival at the camp they suffered from biliary fever (equine piroplasmosis) and sand colic, and it was soon noticed that the precentage of animals rendered unfit after a set of military manœuvres was very high, and that the number which became lame without appreciable cause increased daily. I saw these animals, and found the sick lines full of horses lame in various limbs, many unable to stand even when lifted up, and a number showing the swollen appearance of the nasal and submaxillary bones, while the *post-mortems* and the condition of the osseous structure left no doubt as to the nature of the disease. During the ensuing years cases of this disease became appreciably commoner in the locality. In 1904 Captain Lane, A.V.D., again reports an outbreak of osteoporosis which appeared amongst the mules, donkeys, and horses at the Military Garrison Camp, Middelburg, C.C.

Nature of the Disease.

In diagnosing cases of this disease, care must be taken to discriminate between *osteoporosis* and the much commoner *osteomalacia.*

"The majority of those who have written about osteoporosis have traced the close relationship between these two diseases of the skeleton more from their preconceived opinion respecting their supposed common cause, viz., a deficiency of phosphates in the food supplied, than from any close similarity which they have been able to discern in their course and pathology" (Hutcheon).

Osteoporosis and osteomalacia are distinct and totally different diseases—different in cause, nature, and treatment, and in the *post-mortem* lesions met with in the osseous structures. The cause of osteomalacia is defective nutrition, and the remedy, to remove that defect by supplying the lacking material. In osteomalacia the lesion is a softening of the calcified portion of the long bones; the spaces in the cancellated tissue are full of an oily material (instead of marrow), and the external appearance of the bone is normal while the internal marrow cavity is filled with a jelly-like œdema. Osteoporosis, as I will endeavour to show, is not induced by defective nutrition, and the *post-mortem* lesions in the bones are quite different. Osteoporosis, so far as our experience goes, only attacks the Equidæ (horses, mules, and donkeys); osteomalacia attacks all our domestic animals if the condition of life and care is such as will give rise to it in any of them.

Until my visit to Middelburg Camp in March 1905 I had considered donkeys to be exempt, but there I saw several of these animals showing the distinct enlargements of the bones of the upper and lower jaw usually associated with the disease.

Previous to the outbreak at the Wynberg Camp in 1898 it was rare to meet with such a case in the Cape Peninsula, now cases are of daily occurrence. In 1904 I saw over 156 cases (mostly separate outbreaks), and any veterinary practitioner in this city sees cases every week; from being a pathological curiosity it has developed into a serious menace to the horse-owners of the country.

No class of horses seems exempt from the disease; race horses in training, horses hard at work, fancy riding and driving horses, and unbroken colts running in pasture, all seem susceptible to the disease. It generally attacks animals under seven, and its course varies very much.

Cause.

It is upon this ground that so many veterinarians differ. A great number, who I feel sure are confounding osteoporosis and osteomalacia, or at all events acknowledging a relationship, attribute the cause of the disease to defective nourishment, *i.e.*, the absence of some of the essential constituents from the food, in this case salts of calcium.

The original essay on the subject by Prof. Varnell is responsible for the theory of a deficiency of lime salts in the food, and Prof. Williams says:—"It will yet be discovered that the disease is due to the absence of some essential ingredient of the food, or its presence in such small quantities as to be insufficient for the necessities of the animal, or the presence of some other constituents of the food in such superabundance as to destroy the perfect composition of the aliment as a whole, and thus render it unfit for assimilation by, or so change it as to cause it to become an irritant to, the osseous system."

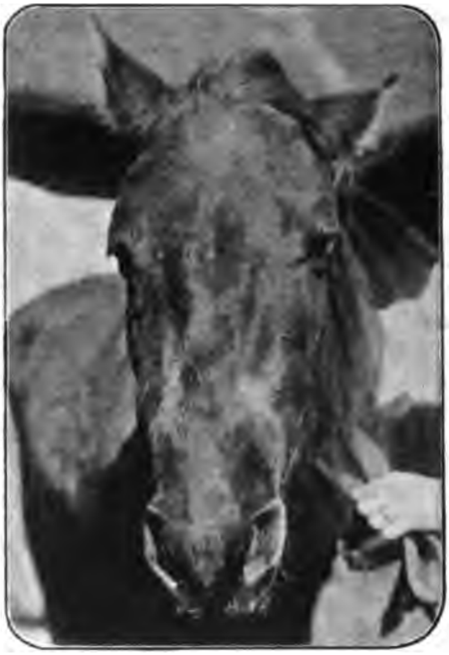

FIG. 1.

Showing the altered appearance of the face caused by the thickening of the superior maxillary bones.

Captain Lane, A.V.D., who has had much experience with the disease, strongly supports this, and attributes the cause of the outbreaks at Wynberg and Middelburg to exclusive feeding on oat forage, particularly that grown upon ground deficient in lime, and considers that cold and exposure may prove exciting causes.

Hinebauch saw cases of ostitis in horses fed on green millet, and fancied this may be at times an important accessory cause.

Harold Elliott (loc. cit.) disagrees with this view, and holds that in

Hawaii the disease only affects animals in certain rainy belts of country. In the case of the last outbreak of the disease amongst the military horses at Middelburg, Captain Lane again states his firm conviction that the disease there was due to malnutrition and exposure, the malnutrition being attributable to the deficiency of phosphates of lime in the oats and hay obtained from certain districts of the Colony.

My observations compel me to regard the disease as being due to some fixed specific contagium. Food and conditions of life seem to have nothing to do with the origin and cause; and, though not directly communicable from sick to healthy animals by inoculation or ingestion, the presence of a sick animal will infect a stable or kraal, and under certain conditions (what these are we do not know) convey the disease to healthy animals put in these stables or kraals.

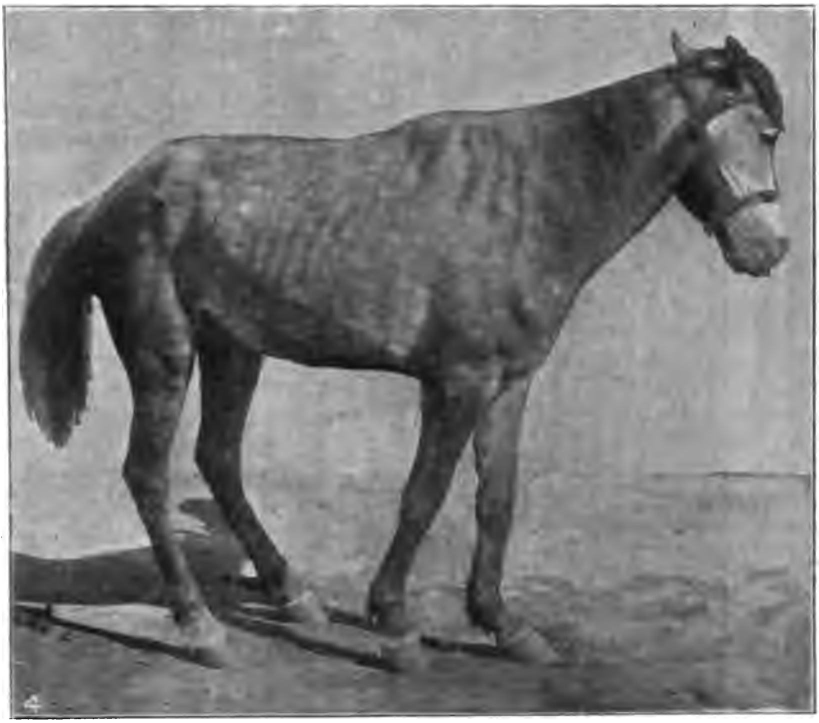

FIG. 2.

Rupture of the perforans tendons and descent of the fetlock joints.

My theory (and it is shared by the chief veterinary surgeon) is supported by a certain mass of facts, and the arguments are more negative than positive. I have seen over 150 cases of this disease in Cape Town during 1904, and the feeding of these subjects was of the most varied character (in several special cases which I will detail at length it will be seen that the ration was abundant and well balanced), while the cases occurred amongst racing horses, private horses kept for pleasure, van and harness horses, and lost wrecks turned derelict on the Cape flats. I had two cases in young fillies running at grass, and twenty-seven in imported animals. When such an array of cases, all at variance with one another, is produced, and when you get the disease indiscriminately attacking all sorts, kinds, and breeds of animals, and animals which exist under all sorts of dietetic conditions, I think to blame the food is false reasoning.

The Cape forage grown in certain districts is actually credited with producing the disease. Some horses have been fed exclusively on Cape forage for the past three generations, and it is only during the past five years that this disease has become so prevalent as to be regarded as of economic importance. Surely if the disease were due to the deficiency of Cape forage in lime, that deficiency would show itself in the crops in the districts where they are grown, but in the very district from which suspected forage is obtained (a district which is famed for its oat crops) we have no record of a case of the disease. And even if we concede the fact that such forage is deficient in certain salts, how is it that this disease has suddenly occurred within the past six years.

Cold, exposure, etc., are said to be assisting or even exciting causes; well, over a hundred of my cases in Cape Town and the suburbs were in stabled, and many cases in rugged, horses. Varnell, though he ascribed the disease to malnutrition, detailed certain cases which, in my opinion, strengthen the contagium theory. "The same owner had two farms not far apart and equal in soil, drainage, and stabling, stocked with horses bred from the same parents, with the same kind and amount of food and work, yet on one farm six cases of osteo-porosis occurred, and on the other not a single case."

M'Neil, in charge of tramway stables in Brooklyn, found the disease destructively prevalent in the best appointed stables and absent from others in the worst hygienic condition, though there was no difference of feeding or management.

Prof. Law, in his *Veterinary Medicine*, gives many instances of the enzootic character of the disease, and states that, in the opinion of Bereus, Hoskins, and other city veterinarians, "outbreaks of this disease occurred quite independently of geological, hydrostatic, or dietetic conditions," and, further, that it was "almost the rule that a fresh horse put in the stall of one that had suffered from osteoporosis soon contracted the disease."

Prof. Law records cases in grain fed animals, on a limestone soil in the city of New York, and the many cases in "Philadelphia and Pensylvania were mostly in animals that had been well fed and were in good condition when attacked" (Marshall). Altitude can have nothing to do with the cause, for we meet it on the seaboard of Port Elizabeth and on the high land of the Witwatersrand.

Infectious or Contagious.

I consider it undoubtedly contagious, though attemps at direct infection have failed, and it may be that an intermediary is necessary for the working out of the life history of the parasite, but the whole evidence connected with the disease points to some definite originating cause. It has been stated how rapidly the disease has increased in frequency and in area of country since the outbreak at Wynberg Camp in (1898), and it is now a well recognised fact in this colony that, once the disease has appeared in a stable, it will continue to manifest itself with more or less frequency in that stable for an indefinite period. Mr Elliott noticed this in Hawaii, and Prof. Law (*Vet. Med.*, p. 581) says, "it is a common experience in Europe and America that a farm or district which has been previously free from the disease suddenly has an outbreak in enzootic form, and this will last for a year or two, then remit, only to appear with its old force after an interval of some years.

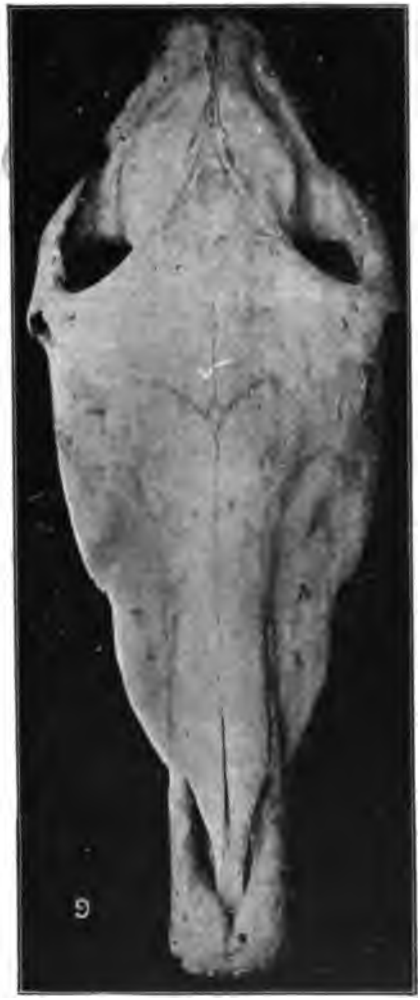

FIG. 3.

Skull from an early stage of the disease.

A merchant in Cape Town, working one pair of horses at a time, has lost in succession three pairs of draught horses and one single buggy horse in two years. Another merchant has in four months lost six animals which were sound when purchased.

The owner of a racing stable placed a two-year-old filly in a stall from which had been taken a case of osteo-porosis; the filly contracted the disease and became a wreck, with all the symptoms and clinical lesions of that disease. This was a curious and instructive case, and one of the most rapid I ever saw. The animal arrived on 21st December, and was then to all appearances perfectly sound; on 24th February I examined her, and, finding her lame, suspected bone disease from the history and surroundings, but could not discern

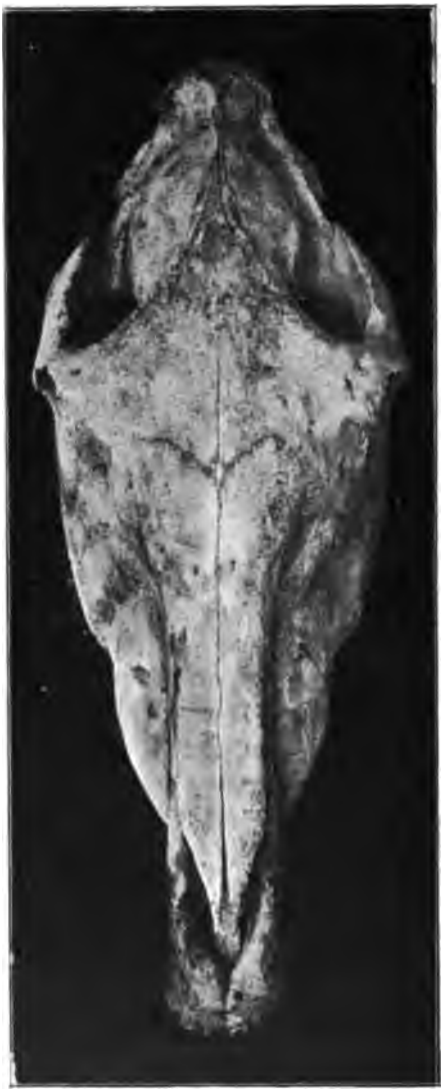

FIG. 4.

Skull showing peculiar erosion of the bones.

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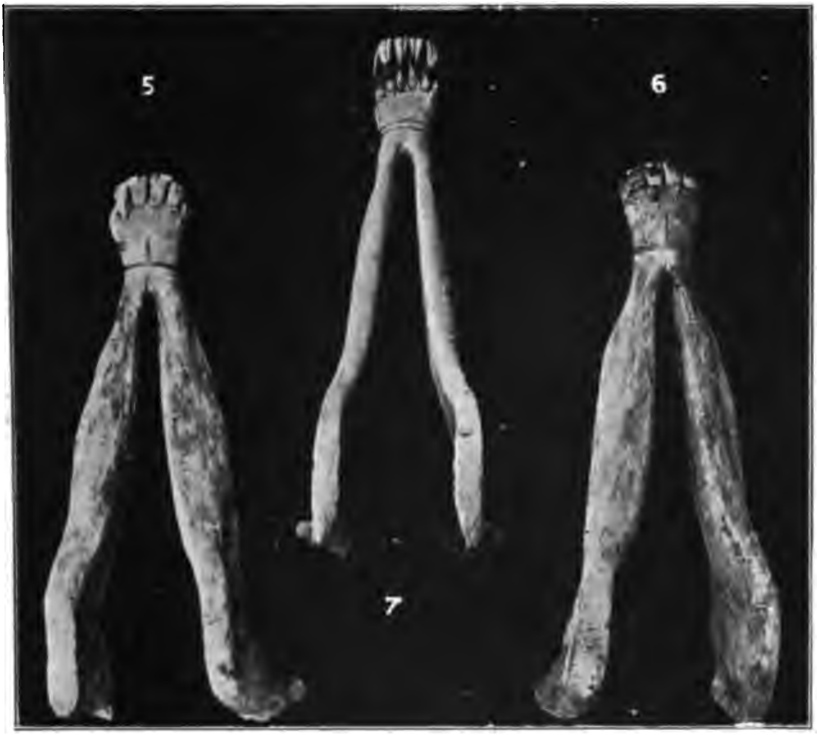
any thickening of the head. On 27th March when I again saw the filly she was dead lame, very poor in condition, and the upper and lower jaws were much swollen and distorted.

I have a stable under observation at present in Cape Town, with seventeen horses in the stud, the work, light pair delivery vans, and the dietetic and hygienic conditions splendid, the manager being a particularly capable horsemaster. Five months ago a horse in that stable was shot on account of osteoporosis, two months ago I killed another, and there are now three more showing signs of the disease. The feed was changed at the appearance of the disease, and bone meal, soluble phosphates, lucerne, imported oats and carrots, with green food were given, but seemed to have no effect on the spread of osteoporosis. The usual feed of this stable is imported oats, bran, and colonial forage, with in winter a few crushed mealies.



FIGS. 5, 6, and 7.

Figs. 5 and 6 show the remarkable thickening of the rami of the inferior maxillæ. Fig. 7 shows the normal lower jaw bones for comparison.

Observant horse-owners in the neighbourhood of Cape Town are perfectly cognisant that the disease "can be brought to a stable," and in many cases can recall the identical animal which brought the infection. Though now common on the Rand, osteoporosis was never seen at Johannesburg until the racing horses went up there from the coast.

All this evidence, these facts and cases, point strongly to the existence of some definite causal agent, although I have failed to discover any special organism in the blood or diseased tissues of an affected animal, and, as I have said, all inoculation and ingestion experiments with animals have failed.

One serious objection to fully accepting the opinion that osteoporosis is due to a specific micro-organism is the fact that the disease can be arrested in its course by changing the patient's surroundings. It may be, however, that certain climatic and local conditions are necessary for the multiplication and development of the hypothetical infective organism of osteoporosis, and there are evidently certain climatic and local surroundings in which it is unable to pass its complete life cycle; especially will this be so if a change of host is necessary (Hutcheon).

Symptoms.

The most common clinical symptoms are fugitive lameness, weakness in the loins, general unthriftiness, and paraplegia with, in the greater majority of cases, swelling of the superior and inferior maxillary bones. In only nine instances out of 150 cases have I failed to find any alteration in these bones.

The symptoms are at first very indefinite and misleading, sometimes simulating rheumatism, and at others muscular sprain; frequently the animal trots short with both hind feet, going on the toes as if there were some lesion of the spinal cord.

In lameness in one limb the pain is manifested when the weight comes on the articular surface, and not when the limb is flexed or extended; frequently both shoulders are involved, rendering the animal immovable. The horse, I fancy, must suffer intermittent fits of pain, as in many cases I have noticed a drawing up of the abdominal muscles and twitching of the flanks, rendering the sternum and costal cartilages (chest line) unduly prominent, as in a case of pleurisy. A limb or some of the ribs and vertebræ are frequently fractured by the ordinary movements of the animal's body. I saw a horse being slung for an injured back break both large metacarpal bones in attempting to rise, and I have seen eleven ribs fractured as a result of another case of the disease slipping down in the stable. Fracture of the vertebræ (either the transverse and superior spinous processes or bodies) has come under my notice in twenty-seven cases, and fracture of the femur in sixteen. One curious case of fracture of the sacrum occurred in casting a stallion which afterwards proved on *post-mortem* to be affected with osteoporosis, and I have seen six backs broken as the result of casting animals affected with the disease to treat them for some other ailment.

In certain severe cases the first symptom noticed was the sudden parting of the perforans tendon at its attachment into the solar surface of the os pedis; it was torn from its attachment, carrying with it a piece of bone the diameter of a sixpence.

In nine cases I have seen rupture or fracture of the sesamoids, and descent of the fetlock (*see* Fig. 1). This occurred at the Experimental Station while an animal (a case of the disease with good head lesions) was being turned round in the stall, the bones parting with a dull crack, distinctly audible. On *post-mortem* it was found that both sesamoid bones of both fore legs were ruptured through their lower third, and the rest of the bones when macerated were of the consistency of biscuit.

It is possible to get cases of osteoporosis without enlargement of the head, but I regard this as one of the most valuable diagnostic symptoms of the disease. Lesions of the limbs and back may simulate rheumatism, strain, etc., but I know no lesion like the osteoporotic head.

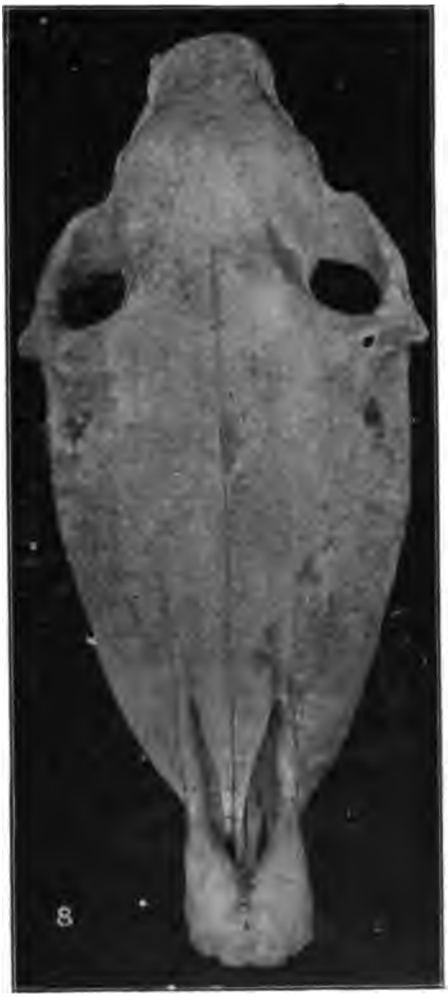


FIG. 8.

Skull from an advanced stage of the disease.

The lesion in the head is generally confined to the bones of the superior and inferior maxillæ; the former are generally swollen symetrically, and the latter are thickened and broad to the touch, the intermaxillary space being very narrow.

LESIONS.

The ribs, vertebræ, and bones of the head and limbs are the most severely affected in this disease.

Varnell gives a perfect description of the lesions in the head bones:—

"In the bones of the head the disease was most advanced in the lower jaw.

"The bone was considerably hypertrophied in its transverse diameter, periosteum readily detachable, and the osseous tissue was of a pink colour, and on pressure a quantity of blood oozed out as from a sponge. The bone was elastic to the touch, and so soft that it could be cut with a knife; the surface of the section had a fleshy appearance and resembled cartilage."

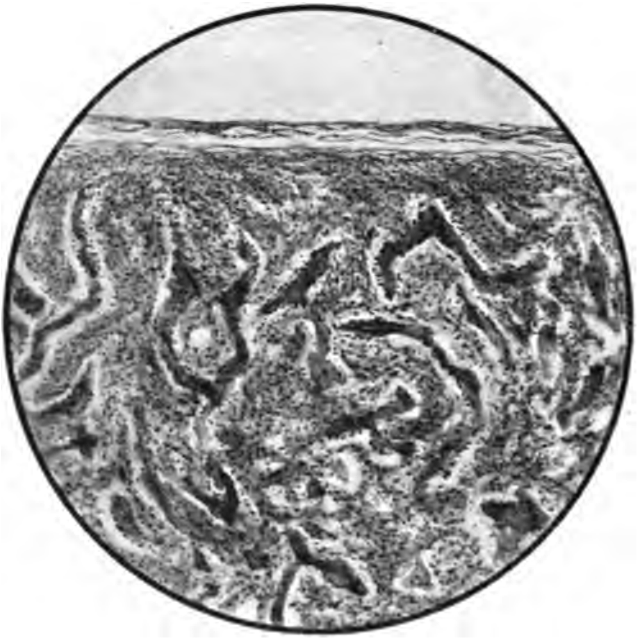


FIG. 9.

Section of diseased lower jaw showing thickening of the periosteum and replacement of osseous by connective tissue.

The frontal and parietal bones in many cases when macerated showed a worm-eaten appearance, resembling the etching action of strong acid. The impression given is that the earthy matter is removed from the bone in discrete areas, which spaces are taken up by connective tissue. The spaces are sometimes as large as a small hazel nut, and portions of this rubber-like connective tissue of the size of dice can be removed, particularly from the inferior maxilla, and hardened and cut in the microtome without previous decalcification or injury to the razor or knife.

In some cases the lace-like structure of the ethmoid bones is completely invaded by this connective tissue, and sometimes a solid column of this new tissue takes the place of the osseous structure in the nasal cavity. In one instance this resulted in the animal dying of suffocation.

This new fibrous tissue is semi-elastic, like indiarubber in consistency, and pale pink and grey in colour, having scattered through its structure small granules of calcium salts and bony spiculæ. In one instance the bones of the head fell to pieces—crumbled into dust—on removal of the animal constituents by maceration. The bones of this head seemed to be composed of earthy matter which crumbled to sand on handling, and the animal had shown very slight distortion of the bones of the head.

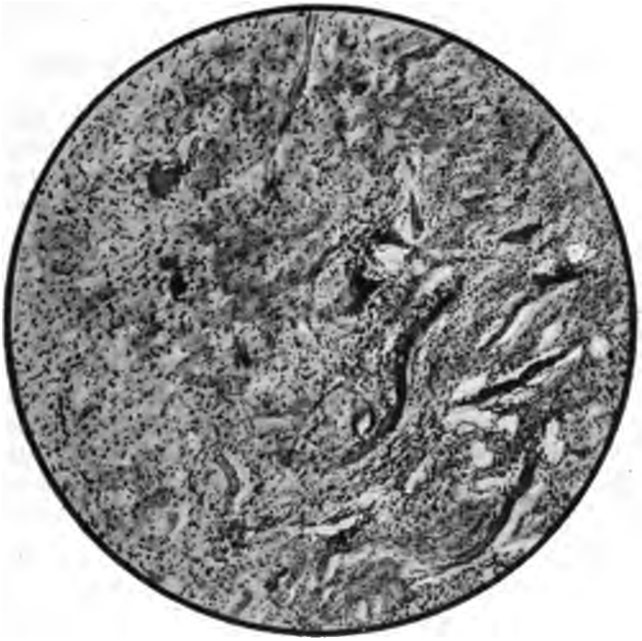


Fig. 10.

Section from lower jaw showing an advanced stage of the disease, in which the normal osseous tissue has almost entirely disappeared.

In a case which was sent up country, while showing bad head lesions, I was fortunate enough to obtain the skull some time afterwards when the animal succumbed to horse-sickness; the swelling of the maxillary bones persisted, but their consistency was dense and firm like normal bone.

The ribs are almost invariably affected, but when macerated out weigh under a third of normal bone, and frequently show signs of fracture. If of long standing the fractured ends are bound together with a tube of fibrous tissue, and there is no attempt at the formation of a bony callus.

I have made over a hundred *post-mortems* on cases of this disease, and select the following as examples :—

Case 1.—A fourteen hands, well built, Colonial-bred six-years-old pony, a family pet, noticed to be sluggish and going lame about three weeks before. When I saw the case the animal was unable to rise, and lay extended, eating with great difficulty, and with a large swollen upper and under jaw. The animal was destroyed, and a careful *post-mortem* made.

The head was the part principally affected, the superior and inferior maxillary bones being much enlarged and softer in consistency than normal; the molar teeth were quite loose in the sockets, and the alveolar septa seemed in a cartilaginous condition; it was possible to cut these bones with a knife, and at the roots of the teeth was much jelly-like matter. When macerated out the skull was extremely fragile, and resembled in consistency a wasp's nest, the bones, temporal, occipital, etc., presenting an eroded and worm-eaten appearance, as if acted upon by strong acid.

The weight of the dried macerated skull minus the teeth was 1 lb. 5½ oz., and with the teeth 3 lbs. 11 oz.

The limbs, vertebræ, and ribs in this case were fairly normal; the articular cartilages of the joints presented an eroded appearance.

Case 2.—A five-years-old Colonial stallion (one of a pair) noticed to be going tender on both fore feet, head and jaws enlarged, and very stiff in back and hocks.

Was kept at the Experimental Station for seven weeks. The lameness in the hind limbs increased, and the cranial enlargement became more pronounced; this animal fed well, but went down in condition in a most marked degree, and sometimes stood for a couple of hours drawing up the abdominal muscles as if in pain.

One day on turning the animal in the stall a sharp crack was heard, and in a few minutes another snap was heard and the horse became immovable; the feet were flat on the ground and the fetlocks almost touching it. The animal refused to move, and when forced to attempt to do so swung its fore quarters forward by standing on its hind legs like a very bad case of laminitis.

On *post-mortem* the two sesamoid bones of both fore limbs were fractured completely across.

This was a good case of the disease. The bones of the body as a whole were more or less affected, and the articular cartilages showed the peculiar eroded patches with a smooth margin; there was no discharge from these patches, which reached right down to the cancellated tissue.

Case 3.—A four-years-old Colonial gelding. Was being attended by a local practitioner in the city, who first suspected azoturea and afterwards slung the animal, diagnosing the malady as injury or disease of the lumbar vertebræ.

The animal struggled a good deal in the slings and at last got its hind feet under it, when a peculiar tearing was heard and the fetlocks descended right on to the ground, and blood began to ooze through the frog and around the coronet at the heels.

On *post-mortem* both sesamoid bones of both hind legs were torn in two pieces, and the bones themselves were so soft that their substance could be rubbed away between the finger and thumb.

This was one of the cases where the symptoms of the superior and inferior maxillary enlargement were absent.

Case 4.—A six-years-old English carriage mare, lame in the hind leg, weak in the back, and generally in a poor and unthrifty condition; was slung as part of the treatment, improved, was allowed to graze in a paddock, and two hours after was found lying down and unable to rise. When killed was found to have fractured the femur at the junction of its upper and middle thirds, and all the ribs on the same side with the exception of the first.

This was a very bad case. The complete pelvis when macerated and dried could be rubbed away between the fingers, and the consistency of the tuber ischii resembled biscuit; the total weight of the macerated innominate bones was under 2 lbs., and each of the dried ribs 2 ozs. The vertebræ cut like a turnip or carrot, and the cancellated tissue was full of liquid red marrow. The superior and inferior maxillary bones were much enlarged, and the teeth loose in their alveoli; the cranial bones were much eroded. The femur could be cut with a knife, and the articular cartilages of the main joints were much eroded.

Regarding the *post-mortem* lesions as a whole, they resemble the disease described in human medicine by the term rarefying ostitis, and that is the exact condition in which the bones appear to be; the solid matter seems lessened, and the tissue resembles a shell which has been steeped in acid.

I have never met with any lesions in the periosteum.

The yellow marrow is generally deficient in amount, and frequently its place in the shaft of a long bone is taken up by red marrow (this may be due to the debility of the latter stage of the disease); the line of demarcation between the yellow and red marrow is very marked, and the entire absence of fat abcut the marrow in particular and the carcase in general is very noticeable.

The erosions on the articular cartilage are numerous, seem non-inflammatory, and reach with clean-cut irregular margins down to the bone tissue; there is no sign of discharge or purulent matter about them.

The alveoli of the teeth are in a gelatinous state, and the molars are loose in their sockets.

The microscopical appearances of sections are much what the *post-mortem* appearances of the bones would lead us to expect, and vary in degree from slight distension of the lacunæ and Haversian canals to complete destruction of the trabeculæ and replacement of all osseous structure by new connective tissue. It would appear as if the new tissue destroyed the original bone.

Curability and Treatment.

I have never seen a case of recovery in an animal kept in the area where it contracted the disease, no matter how treated. I have at present under treatment at owners' stables and at the Department's experimental stables cases of this disease which were placed under care and treatment as soon as the slightest signs of osteoporosis were noticed, and given the most nourishing food, supplemented by liberal rations of bone meal (containing sufficient phosphates to supply the bodily wants independent of the proportion of these salts in the food), and so far the results from a curative or palliative point of view are negative.

Captain Lane recommends changing the dieting to oats, 6 lbs.; bran, 1 lb.; clover lucerne, 8 lbs.; salt, ½ oz.

From the figures in Captain Lane's Report this treatment has been a great success; out of 335 mules affected, 249 were cured, 53 were relieved, 25 died, and 10 remained. The cured, I was given to understand, were working daily or as required, and no relapses had been noticed.

This has not been our experience in the Colony.

Cases of natural recovery or arrest of the disease occur with considerable frequency when an animal in the initial stage of the disease is removed to another district. Colesberg horses if they contract the disease in Cape Colony recover when taken back to their own veldt, but we may generally take it that the thickening of the superior and inferior maxillary bones persists.

Amongst the drugs which I have tried without avail are phosphates of lime, potash, and soda, bone meal, pot. iodide, salicin, salol, phenacetin, phosphoric acid, and tonics. Locally, blisters and the thermo-cautery.

I also tried the treatment recommended by Cary, viz., intravenous injections of barium chloride once a week for four weeks, and ½ oz. doses of sodium salicylate thrice a day.

The following are the statistics of ninety cases:—

Lame in hind legs	30
Lame in fore legs	12
Lame hind and fore	9
Lame back	20
Lame back and hind limbs	15
Lame back and fore limbs	4
	90
Showed head lesions	86

Summary.

1. The disease was unknown or very rare in this colony twenty years ago.
2. It is on the rapid increase.
3. It is contagious.
4. It is not due to dietetic causes.
5. Altitude, age, and breed have no bearing on the prevalence of the malady.
6. Within certain areas the disease has become enzootic.

BRONCHO-PNEUMONIA OF CATTLE: ITS ASSOCIATION WITH BACILLUS BOVISEPTICUS.[1]

By PAUL G. WOOLLEY, M.D., Director of the Serum Laboratory; and WALTER SORRELL, D.V.S., Veterinarian, Serum Laboratory.

From the Government Serum Laboratory, Manila. P. I.

THE reason we care to dwell upon these cases is not that the above-mentioned pulmonary affection is uncommon or not well known, but because of its relation, here at least, to hæmorrhagic septicæmia.

It is agreed by all authors that the causes of catarrhal pneumonia, in animals as well as in man, are not always the same. However, in general the chief ones are also those of acute bronchitis; following this it happens, either because of swelling of the mucous membrane, or by aspiration of bronchial secretion, that the small subdivisions of the bronchi became plugged, and a condition of atelectasis arises in that part of the lung supplied by the affected bronchioles. Then, because of the *locus minoris resistentiæ* furnished by the atelectatic part, and because of the usual presence of infectious material in the bronchial secretions and in the aspirated material, inflammatory processes occur, which may be limited or which may spread by continuity to the neighbouring tissue. The changes following atelectasis may be either a gradual atrophy of the affected parts of the lung, or, following infection, bronchiectasis, abscess formation, pleurisy, gangrene, etc., resolution, or calcification. The disease occurs especially in young animals and in older ones whose physical condition is below par. In young animals it may be epizoötic.

The symptoms are said to vary. Schneidemühl says that the first signs point to acute bronchitis, upon which ensue pulmonary symptoms, such as increased respiratory frequence and cough.

The course of the disease, according to Dieckerhoff and Schneidemühl, is neither typical nor regular. Sometimes it progresses very rapidly, and in other cases it may last for weeks or months.

In the more chronic ones a purulent pneumonia with pleuritis and pulmonary gangrene may ensue and death follow. It is to this last group, that of chronic cases, that ours belong.

These occurred in the herd of the Government Serum Laboratory. The calves were imported for use in the preparation of vaccine virus. The cattle were used in the preparation of anti-rinderpest serum. All of the calves originally came from China, and upon arriving in Manila had been treated with prophylactic doses of anti-rinderpest serum and kept under careful observation for several weeks before they were used for the intended purpose. At the time of vaccination there was no indication of disease in any member of the herd; their appetites were good, they were in excellent physical condition, and their temperature curves were normal. (Inasmuch as tuberculosis has never been observed in cattle here, the vaccine calves are not slaughtered after collecting the virus, but are kept on hand until disposition can be made of them in some other way. As a rule, the physical condition of the animals improves after taking the vaccine. When this is not the case, slaughter is resorted to—when there is no particular reason, as in the present instances, for allowing the animals to live.)

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[1] Read at the Second Annual Meeting of the Philippine Islands Medical Association 1st to 4th March 1905.

The steer mentioned below was also imported from China for use in the preparation of anti-rinderpest serum, but its condition was never satisfactory for this purpose.

The histories follow:—

Case I.

Calf No. 437 was received at the laboratory on 12th January 1904, and received 100 cc. of anti-rinderpest serum at once. It remained apparently well with a normal temperature until 24th January, when the latter rose to 40·7° C. The next day the temperature was 40·8° in the afternoon, and the following day it reached 41·2°. From this point it gradually declined to normal on 30th January. On 1st February 5 cc. of virulent rinderpest blood was given to the animal, and on the third day after it was vaccinated. Following this operation, there was what at first appeared to be the usual temperature of vaccinia, which reached 40·6° on the fifth day after vaccination. But this temperature, instead of declining, persisted with slight variations between 39° C. and 41° C. until 1st March, when it became normal, and stayed so until death, 24th March 1904. On several occasions the blood of the animal was examined for trypanosomes with negative results.

During the course of the disease the animal gradually lost weight in spite of a reasonably good appetite, and as it progressed the appetite became impaired and the coat rough and staring. There was no cough, nor in fact any other notable symptom than the gradual wasting.

The autopsy was made about twelve hours after death. The only appreciable lesions present were in the lungs. The other organs were in an apparently healthy condition, with the exception of the prescapular lymph glands, which were enlarged and œdematous. The apices of the lungs were chiefly affected. These portions of the organ were almost completely solidified. The surfaces of the hepatised portions were mottled with yellowish-white areas which stood out distinctly upon a reddish-purple ground. In palpating, the finger was able to detect that these lighter areas were firmer than the darker coloured portions of the lungs, which had an œdematous consistency. Over several of the nodules there was a thin fibrinous membrane, under which the pleura was congested and roughened. Upon section, the affected portions of the organs cut with increased resistance, and from the incisions a frothy serum oozed. The general colour of the surface was dark red, mottled by the greyish-yellow of the sometimes almost caseous nodules. Sections of the latter had all the microscopic characteristics of the grey hepatised tissue of pneumonia, being granular and dry. In an occasional one the process had gone beyond the simple hepatisation, and the centre had softened, producing a rather creamy material.

From several of such tubercles cultures were made upon agar and in bouillon, and pieces of the tissue were placed in Zenker's solution and absolute alcohol, for further study. Smears made from the pulmonary nodules showed a few small ovoid bacilli, which stained with the ordinary anilin dyes and were decolorised both by Gram's and Gabbett's methods.

Cultures of organisms were obtained which had all the characteristics of the bacillus described by one of us as the cause of an epidemic of hæmorrhagic septicæmia among the carabao of the Government corrals during the past year. The chief characteristics of this organism were its polar staining, rounded ends, non-motility, and occasional encapsulation. It grew invisibly on potato, did not produce gas in either solid or liquid glucose media, did not coagulate or acidify milk, did not form spores or liquefy gelatin, but did reduce nitrates and also gave the indol reaction. It was pathogenic for guinea-pigs, causing death within twenty-four hours after intraperitoneal injection of 1 cc. It was the bacillus bovisepticus of Kruse.

The tissues preserved for sectioning were imbedded in celloidin, and sections from these were stained with hæmatoxylin and eosin, by Gram's method, and by the carbol-fuchsin-acid one for tubercle bacilli. The stained sections showed a generally œdematous condition of the parenchyma. The trabeculæ were somewhat thickened, and the fibrous tissue of the affected portion of the lungs was generally increased. The epithelial linings of the bronchi were convoluted and hyperplastic, the cells being in many places three or four layers thick, and in the dilated lumina there were considerable accumulations of epithelial cells, polymorphonuclear leucocytes, and a minimal amount of fibrin.

The air spaces of the affected lobes which were not involved in the consolidation were filled with a granular material, with which there were occasional desquamated cells and leucocytes. The peri-bronchial tissue was the seat of well-marked round-cell infiltrations, and the peri-bronchial connective tissue was considerably increased.

Sections of the small nodules of consolidation showed that the chief change in these was a coagulation necrosis. The centres of these areas were crowded with leucocytes and granular, cellular detritus enmeshed in fibrin. The walls of the air cells were perceptible as faintly pink-stained bands, in which no nuclei or only shadows could be seen. As the periphery of these areas was approached the lines became more distinct, and about the latter was a zone of congestion. There were no giant cells seen in any sections, and no acid-fast bacilli could be found. There were, however, a number of very short bacilli between the cellular contents of the abscesses.

Case II.

Calf No. 423 was received from Hong Kong on 6th January, 1904, and given a preliminary prophylactic dose of 100 cc. of anti-rinderpest serum. It was vaccinated on 13th January, after which its temperature rose to 40° C., where it remained with slight remissions until 19th January, reaching normal on the 20th. On 21st January it received 10 cc. of virulent rinderpest blood, and following this the temperature again rose and varied between 39·2° and 40·6° for the next eight days. On 1st February it received 50 cc. of virulent blood, and following this there was again a rise, which, however, was transient. From this time on the temperature remained within normal limits. Death occurred on 30th April, somewhat more than three months after the arrival of the animal at the laboratory.

During the last month or five weeks of its life a steady and gradual decline was evident. The animal lost weight in spite of a constantly fair appetite. Upon several occasions the blood was examined for trypanosomata, each time in vain. No cause could be found for the wasting. There was no cough, in fact, no other symptoms than the gradual emaciation and increasing weakness.

The day before death the animal was unable to stand, but was lying in its stall in the hospital shed, eating the grass before it. It died the following night, and was found in the morning in its sleeping position.

Upon opening the thorax a considerable amount of clear serofibrinous fluid gushed out. The peritoneal cavity contained no fluid and was apparently normal. The subpleural and mediastinal tissues were œdematous and gelatinous in appearance. The prescapular glands and periglandular tissues were also œdematous. The liver appeared pale but otherwise normal. There were a few petechiæ in the capsule of the spleen, which was of normal size and consistence.

The cæcum contained a number of strongyles and flukes. There were no gastro-intestinal hæmorrhages or ulcerations.

The lungs were the seat of a patchy hapatisation, which involved about one-half of the entire pulmonary tissue, chiefly the apices and anterior lobes. In the hepatised areas, and perceptible to the eye and finger, upon the surface of the lungs, were small nodules. These were firmer and paler than the adjacent tissue, which had an almost purple colour. Section of these organs showed that the fibrous tissue was generally increased in the affected portions, the trabeculæ being quite prominent. The surfaces of cut sections showed mottled grey and red, the grey being most apparent in the centres of the lobules. Certain of these grey areas were quite dry and almost caseous, and were as large as a navy bean.

From these nodules cultures were obtained on agar of a non-motile polar-staining bacillus, which did not stain by Gram's method and did not form spores. The colonies in agar varied in size from 0·5 to 1 mm. in diameter, were thicker and opaque at their centres, were not chromogenic, had smooth regular edges, and were somewhat sticky and mucilagenous. On agar slants after twenty-four hours the growth was in the form of colonies, but with a tendency to confluence. The water of condensation was clouded.

On glycerine-agar the growth was more luxuriant, quite thin but almost opaque, and so moist that it was inclined to run down the surface of the slant. Milk became just perceptibly acid after seventy-two hours, but without coagulation or reduction of the litmus. The growth on potato was invisible. In glucose broth and agar no gas was formed. In Dunham's peptone, to a litre of which 5 cc. of a five per cent solution of potassium nitrate had been added, a brilliant cholera-red reaction could be obtained after twenty-four hours.[1] After seventy-two hours a pellicle appeared in the ordinary bouillon; the medium was diffusely clouded, and a flocculo-gelatinous sediment was thrown down.

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[1] This method was suggested to us by Dr. W. B. Wherry. For some time we have all been troubled by the inconsistency of the peptone used in making Dunham's solution. Dr Wherry, however, found that a constant reaction could be obtained by adding traces of nitrates to that fluid.

Of a twenty-four old broth culture, 1 cc. was injected into the peritoneum of a healthy guinea-pig. The animal survived this treatment.

Celloidin sections cut from material fixed in Zenker's solution, and stained with hematoxylin and eosin, showed changes that correspond in all general respects with those described in the previous case.

Case III.

Calf No. 415 was received at the laboratory on 29th December, 1903, and given the usual prophylactic dose of 100 cc. of anti-rinderpest serum. The temperature remained normal until after vaccination, which was done eight days later. Following this, on the third day, the temperature rose abruptly to 41·6° C. and then gradually fell to normal within the next week. On 20th January 1904 it was given a subcutaneous injection of 10 cc. of virulent rinderpest blood, following which there was a reaction of 1·4° C., after which the temperature fell to normal and continued so to the time of death on 14th May 1904, almost five months after being received. At no time were trypanosomes found in the blood. The clinical history in the case was not unlike that in the previous one; the salient points were gradual loss of weight in spite of retention of appetite, normal temperature, no cough, roughened and staring coat, and loss of blood.

At autopsy the chief lesions, and, in fact, the only macroscopic ones, were in the lungs. There was not as much œdema as in the previous case, and there was a less general pulmonary involvement. But here, as in the other ones, the apices and anterior margins of the lungs were partially solidified, mottled with red and grey, and filled with small firm nodules, some miliary and some the size of a large bean. Some of these on section proved to contain a semi-fluid purulent material, while others were dry and grey.

Cultures were made as before from these nodular lesions, and in this case two organisms were isolated, one B. pyocyaneus, the other a polar-staining bacillus agreeing in general with the organism previously described, but varying from it in some cultural characteristics.

Morphologically, it was identical with the organisms from the previous cases. Culturally, variations were most marked in broth and on agar. On the latter, the growth was generally composed of colonies, but these were somewhat larger than the ones described for B. bovisepticus. The growth was much more luxuriant and whiter, and there was a decided tendency to confluence on the part of the colonies.

In bouillon there was at first a diffuse cloudiness with no sediment and no pellicle. Later, a gelatinous sediment was deposited, a very delicate pellicle was developed, and the body of the medium became clear. There was also a deposition of flocculi on the sides of the tubes.

The cholera-red reaction was obtained after twenty-four hours. Milk was unchanged, and there was an invisible growth on potato.

Intraperitoneal injection of 1 cc. of this organism killed a healthy guinea-pig in less than twenty-four hours. At the autopsy an acute hæmorrhagic peritonitis was discovered, and the organism was recovered.

Case IV.

Calf No. 464 was received at the laboratory on 12th January 1904, and was injected with 100 cc. of anti-rinderpest serum. The following day its temperature was 40·1°, and on the succeeding it was normal and remained so for the next ten days. On 20th January it was vaccinated with vaccine virus, and following this its temperature rose, reaching a maximum, 41·1°, on the day after the vaccine was collected. Two days later it was again normal. On 1st February 1904, 10 cc. of virulent rinderpest blood was injected subcutaneously, and following this the animal's temperature became quite irregular, reaching 40·5°, and with daily remissions of 1 to 2 degrees; but after twelve days the maximum had fallen to about 39·2° C. and with smaller remissions. This continued until death occurred, 9th May 1904, about four months after its arrival in Manila. Up to this time it was able to eat, but it gradually became emaciated and very weak.

At the autopsy the lungs were found to be affected in a manner similar to the condition of the others described above. In this instance, however, the apices were alone affected over one area, each of about the size of the palm of a hand. These were purplish-red, œdematous, and contained small areas of solidifaction, which appeared greyish or grey in sections. There was a fibrinous exudate over the pleural surface of some. The other organs showed no change other than mild parenchymatous degeneration. The body was anæmic.

Cultures were made from the nodules in the lungs. On the original plate cultures, two types of colonies were noted; one very small, similar to the typical colonies of B. bovisepticus, the other larger and with more tendency to spread, and upon slant cultures to coalesce. Both of these were studied.

The only cultural differences in these two strains were noted in agar, upon which one showed a greater inclination to spread and coalesce, and in broth, in which one (464^1) caused a uniform clouding with a gelatinous sediment, while the other (464^2) clouded the media less diffusely and formed flocculi, which were present not only in the body of the fluid, but also on the sides and bottom of the tube. Both gave a brilliant indol reaction. Both were pathogenic for guinea-pigs.

Sections from material fixed in Zenker's solution and imbedded in celloidin showed the same histological picture as that described in the above cases.

Case No. V.

Calf No. 491 was received at the Serum Laboratory on 24th February 1904, and was given a prophylactic dose of 100 cc. of anti-rinderpest serum. A transient rise of temperature to 41·2° C. and a gradual return, during seven days, to normal followed this. On 5th March 1904, 5 cc. of virulent rinderpest blood was injected subcutaneously, upon which a scarcely perceptible rise of temperature lasting but twenty-four hours was noted. Four days later the animal was vaccinated, and again the temperature rose, remained between 39° and 40° C. during the inoculation disease, and then gradually fell to normal. On 25th March 1904, a second injection, this time 50 cc., of virulent blood was made, and this gave rise to a slight increase in temperature, which lasted but forty-eight hours.

During the following month the temperature remained above normal, varying from 39 to 40° C., and only dropped immediately before death. The animal died on 5th May 1904, a little over two months after its arrival at the laboratory.

At the autopsy, done about eighteen hours after death, the upper halves and the anterior lobes of the lungs were found to be of mottled purplish-red colour, and almost completely consolidated. In the areas of consolidation were a few nodules, generally of a pyramidal shape with their bases in the pleura.

The pleural surfaces of these were covered with a thin fibrinopurulent exudate, which upon removal showed the yellowish colour of the nodule. On section the nodules appeared to be abscesses, surrounded by consolidated pulmonary tissue, and containing a thick yellowish pus. Throughout the consolidated portions of the lungs there were smaller, almost miliary, firm areas. In general these were in the centres of lobules about which the trabeculæ surrounding them were increased in size and quite prominent. The other organs showed no marked changes.

Cultures from the abscesses upon glycerin-agar and in broth showed no growth.

Histological examination of sections showed approximately the same arrangement in and about the nodules as in the previous cases.

Case VI.

Calf No. 453 was received at the Serum Laboratory on 12th January 1904, and given 100 cc. of anti-rinderpest serum. The next day its temperature was 40·2° C., after which it fell to normal, remained so for three days, and then gradually rose to 40·8°, afterwards again gradually falling, the course covering a period of one week. From this time until death the temperature remained normal, except for a transient rise after vaccination, and one following injection of 50 cc. of virulent rinderpest blood. It died on 27th April 1904, about three-and-a-half months after coming to the laboratory.

At the autopsy the pulmonary lesions were similar to those found in No. 491, though less extensive, being almost limited to the apices and the extreme exterior borders.

Plate cultures from the nodules after twenty-four hours at 37° showed three slightly variant types of colonies. Transplants were made from each type and studied as B. 453^1, 453^2, and 453^3.

After carefully comparing their appearances in and on various media, no other difference could be seen than that of size. B 453^1 was a trifle larger, generally, than the other two. All produced typical colonies, *i.e.*, the ones which were larger, more opaque, and which showed a slight tendency to coalescence.

These various differences between these strains will be discussed later. Suffice it to say now, that, in general, the organisms in their cultural and morphological characteristics were identical with B. bovisepticus.

Histological examination showed a condition that agreed with that seen in previous cases.

Case VII.

Steer No. 383 was received at the Serum Laboratory on 6th January 1904, and was given 100 cc. of anti-rinderpest serum. Two days later it showed some indisposition, and upon examination the temperature was found to be normal, the pulse somewhat accelerated, respirations rapid and shallow, coat staring, appetite diminished and rumination performed with indifference. Later the temperature became irregular, with an evening rise which often reached 40° C. About ten days after the first examination the animal developed a shallow, painful cough, which became more marked as the disease progressed. Tuberculosis, although up to that time unknown in these islands, was suspected, and the steer was killed, sixty-one days after coming to the laboratory.

At the autopsy a very widespread pathological condition of the lungs was found, affecting almost the entire extent of both organs. This consisted in a chronic bronchitis with bronchiectasis upon which a broncho-pneumonia had been engrafted. The bronchial walls were injected and covered with a sticky muco-purulent material. There were several bronchiectatic cavities, one the size of a goose egg, the others smaller, which had smooth fibroid walls within which were greenish-yellow muco-caseous masses of very foul smelling material. The parenchyma of the lungs was studded with small areas of consolidation, some of which had undergone softening. The lungs were generally œdematous. There were no marked changes elsewhere save congestion and cloudy swelling.

Cultures made from the small nodules at the time of autopsy showed no growths.

Tissues were preserved in Zenker's solution and embedded in celloidin. Sections from these materials were stained with hæmatoxylin and eosin, thionin, gentian violet, following the Gram and Weigert methods, and by the ones used to demonstrate tubercle bacilli.

The lungs on section showed an intense peri-bronchial infiltration, which was composed for the most part of cells of an epithelioid character with abundant protoplasm and vesicular nuclei. There was an increase in the connective tissue of these accumulations, which were also more or less regularly enclosed by fibrous capsules, making it appear as though the epithelioid accumulations had occurred between the mucous membranes and the surrounding muscular layers. Outside of the encapsulating layers were occasional lymphoid accumulations.

The mucous membrane of the bronchi was hypertrophied and corrugated, the cells being two or three layers thick and many of them vacuolated. The lumen was filled with mucous material in which numerous leucocytes were embedded.

In several places the mucous membrane on one side was hypertrophic, while on the other it was flattened and atrophied. Projecting into the lumen from the hypertrophied side were masses composed of small round cells and stroma, about whose internal edges were a row of large cells of peculiar appearance. One of these was an enormous cell whose protoplasm was very large in amount and whose nucleus was about three to four times the size of a poly-morphonuclear leucocyte, and vesicular. The others were not so large, but were of the same type, except one large giant cell from whose position, and the arrangement, it was plain that it had been formed by the coalescence of epithelioid (endothelial) cells. The remainder of the lumen was filled with cells similar to those described.

That the structures of the growth were granulomata might be seen in this same section, for the mixture of round epithelioid cells projecting into one large bronchus was well supplied with mature and immature blood vessels. There was no giant cell formation in these epithelioid or round-celled nodules. The only place, apparently, where this was found was on the lumen side of the projecting growths, and even here the occurrence was an exception.

The trabeculæ of the lungs were increased in volume. The perivesicular connective tissue was increased and the parenchyma was injected.

It certainly seemed that in some places there was a tendency to syncytial formation in the mucous membranes of the bronchi, especially in those in which the peripheral growth had encroached largely upon the lumens.

In other sections the profuse fibroid overgrowth was most well marked. In such bronchi, so nearly obliterated that nothing but remnants of mucous membrane were to be seen imbedded, masses of round and vesicular cells occurred. Giant cells could be seen in these, but here, too, these were nearer the peripheral margin of the nodules, and they seemed in a certain degree to have some relation to the smaller bronchioles upon which the cellular accumulations had encroached.

In other sections nodules could be seen whose centres evidenced commencing necrosis.

The liver showed a remarkable degree of fibroid changes, evidently originating about the bile ducts. The tissue, besides its formative elements, containing many cells with vesicular nuclei. New gall-duct formations were also noticed.

In the sections no acid-fast organisms were discovered.

Cases VIII. and IX.

Calves Nos. 486 *and* 405.—Neither in their clinical symptoms nor in their general condition did these animals show any marked symptoms of disease. Their temperature kept within normal limits except after virulent blood injections and after vaccination. They had no cough. They ate up to the day of death.

In the case of No. 486, which became much weaker than did No. 405, appetite was present and the animal ate its fodder eight days after it had been unable to rise to its feet.

The lesions were comparable to those described in the other calves, but in both cases they were limited to the apices of the lungs. From the lesions of each animal an organism was obtained in cultures which was similar to the one already described, viz., to B. bovisepticus.

The histological examination, while the processes were less advanced, showed approximately similar changes.

That these cases arose in the way Dieckerhoff and Schneidemühl describe, there can be but little doubt. In certain animals, whose physical condition warranted slaughtering, we have noticed small areas of atelectasis, which might or might not have been congenital, a point which we have been unable to determine. In certain others we have found both acute and subacute bronchial changes, with no macroscopic parenchymatous changes. Whenever two such conditions coincided in the same animal the ideal opportunity would be

Pellicle.	*Broth Sediment.*	*Fluid.*	*Side of Tube.*	*Gelatin Liquefact.*	*Agar.*	*Milk.*	*Potato Growth.*	*Glucose Bouillon.*	*Peptone Indol.*	*Gram's Stain.*	*Motility.*	*Pathogenesis.*	*Remarks.*
Scanty	Flocculo-viscid	Diffusely clouded	Precipitate	0	Large colonies. Tendency to confluence	Nochange	Invisible	No gas	Pos.	0	0	Guinea-pig	Medium size. Indol reaction brilliant
Do.	Do.	Cloudy flocculi	Do.	0	Fine dew-drop colonies. No confluence	Ibid	Ibid	Ibid	Do.	0	0	Do.	Small indol reaction, brilliant
Whitish	Do.	Diffusely clouded	—	0	Moderately luxuriant, confluent growth	Very faint acid	Do.	Do.	Do.	0	0	Do.	Largest of nine races
Scanty	Do.	Clear	Precipitate	0	Confluent growth	Nochange	Do.	Do.	Do.	0	0	Do.	Medium size. Indol reaction brilliant
Whitish	Viscid	Diffusely clouded	—	0	Very delicate growth of fine colonies. No confluence	Ibid	Do.	Do.	Do.	0	0	Do.	Small
Do.	Do.	Do.	—	0	Delicate growth of small colonies. No confluence	Do.	Do.	Do.	Do.	0	0	Do.	Very small
Do.	Do.	Do.	—	0	Delicate growth of small colonies. No confluence	Do.	Do.	Do.	Do.	0	0	Do.	Do
Scanty	Do.	Clear	Precipitate	0	Small colonies. No confluence	Do.	Do.	Do.	Do.	0	0	Do.	Do.
Do.	Do.	Do.	Do.	0	Very small colonies. No confluence.	Do.	Do.	Do.	Do.	0	0	Do.	Do.

It may be well to note here that, unless otherwise stated, the reaction of the media used is 1 per cent. acid to phenolphthalein, and it was brought to this degree not by neutralising with alkali and then adding hydrochloric acid, but by adding simply enough alkali to produce the right degree of acidity.

An excellent discussion of the cultural characteristics of the members of the bacilli of hæmorrhagic septicæmia will be found in Moore's book on "The Pathology of Infectious Diseases of Animals."

afforded for the production of more serious pathological complex, and we imagine this is exactly what has happened in certain of these cattle.

It is an interesting feature of these cases that the bacilli of hæmorrhagic septicæmia should be associated with so large a proportion.

Just what the duration of the disease has been in these animals can not be determined accurately. In the steer it was undoubtedly at least fifty days, and in the others it was probably thirty to ninety days.

Infection had undoubtedly taken place in each case following the primary bronchitis, perhaps during the later stages of pneumonic change.

Since the organisms present in the lesions proved to be the bacillus of hæmorrhagic septicæmia, we may infer that this is a common inhabitant of the respiratory tract of animals here, as it is in the United States.

Further, we may conclude from the above-mentioned facts that these cases are not examples of chronic infection with B. bovisepticus, but simply of an implanted infection in the course of other pathological conditions, or, perhaps in certain cases, of a terminal infection. It seems probable, from our experience, that when primary infection with this organism occurs the disease runs a very acute course.

Another interesting fact was brought out in the bacteriological investigation of these cases, namely, that the cultural characteristics of the B. bovisepticus are not constant. Even with organisms of approximately equal virulence the growths on and in media vary considerably (these are perhaps most marked in bouillon, as the accompanying table will show), and with races of unequal virulence the differences are still greater.

A fuller discussion of the variations of B. bovisepticus, especially as regards pathogenicity, must be left for further study. From the few facts at the present time available we are not willing to draw conclusions.

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THE TEMPERATURE OF HEALTHY DAIRY CATTLE.

By G. H. WOOLDRIDGE, M.R.C.V.S., Professor of Medicine, Royal Veterinary College of Ireland.

I HAVE been impressed on many occasions, when referring to the works of various observers, by the considerable differences in the average temperatures of apparently healthy cattle quoted by them, and also by the variations one might expect to meet with during health. Consequently, I determined to make observations with a view to ascertaining, as far as possible, the mean temperatures of healthy cattle taken per rectum, and the extremes between which they might be expected to fluctuate under normal conditions.

Robertson[1] made observations on 352 cows and oxen, and gave 101·9° F. (38·85° C.) as an average, with extremes of 100° and 104·5° F.

Singleton[1] gives an average of 101·5° F. (38·6° C.), with extremes of 100° and 103·3° F. His observations number 100.

Hobday[1] made observations on eighty-seven cows, and agreed with Singleton in the case of the mean temperature, viz. 101·5° F.; but his range is a little lower, 99·5° F. to 103° F.

Fred. Smith[2] gives 101·8° F. to 102° F. (38·7° C. to 38·8° C.). Colin,[3] Meade Smith,[4] and Thanhoffer,[5] all give identical figures, viz. 100·4° F. to 101·3° F. (38° C. to 38·5° C.).

Friedberger and Fröhner[6] give 101·8° F. (38·8° C.) as the average, and quote Hadschopulo, who took 50,000 temperatures at Moscow, and gave an average falling between 101·1° F. and 101·8° (38·4° C. to 38·8° C.).

Thanhoffer[7] also quotes Gavarret-Rosenthal, who states the very low average of 99·5° F. (37·5° C.).

On comparing these figures it will be seen that Robertson and Fred. Smith give a *mean* temperature of 101·9° F., which is ·6° F. higher than Colin and others give as the *higher extreme*. Again, comparing the averages of Robertson and Gavarret-Rosenthal, we find a difference of almost 2·5° F., the former giving 101·9° F., and the latter 99·5° F.

The observations I have recorded were all made on apparently healthy dairy cattle. They number 1395, and were made on 174 animals. I shall presently draw attention to the fact that a large proportion of these animals were subsequently submitted to the tuberculin test, and that many of them reacted. But it is advisable first to see what their average temperature is, in order to compare it with those quoted by previous observers who omit to state whether the subjects of their observations were proved free from tuberculosis, in which case I think it is fair to conclude that they come under the same heading of "apparently healthy" animals. I find, then, that the average temperature of these 1395 observations is 101·6° F. (38·66° C.), which is ·1° F. higher than Hobday and Singleton give, and ·3° higher than Colin and others give as the higher extreme.

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[1] Quoted by Schäfer, "Text-book of Physiology," p. 790

[2] "Veterinary Physiology," p. 306.

[3] "Traité de Physiologie Comparée des Animaux, vol. i., p. 1046

[4] "Physiology of the Domesticated Animals," p. 696.

[5] "Vergleichende Physiologie und Histologie," pp. 476, 477.

[6] "Lehrbuch der Klinischen Untersuchungsmethoden," p. 166.

[7] *Op. cit.*, pp. 476, 477.

Much greater importance, however, must be attached to the statistics based on observations made on animals proved to be free from tuberculosis, which is often responsible for considerable fluctuations. I know of no such observations previously recorded. In fact, nearly all the statistics we have were compiled before tuberculin was introduced into regular practice; and its use is the only definite method we have of detecting most of the early cases of the disease.

Of the previously mentioned animals, 63 of those tested failed to react to the tuberculin, and were consequently considered to be free. On those 63 animals I have 520 observations; and the average temperature I find to be 101·4° F. (38·5° C.), The lowest recorded temperature of the 520 was 100·4° F. (38° C.), and the highest observed was 102·8° F. (39·3° C.); but these extremes were very rarely indeed met with. The lowest average of any individual was 100·8° F., and the highest average was 102·2° F.

Of the 63 healthy animals, only 7 had an average temperature below 101° F.; and the same number had an average temperature of 102° F. or over.

Thus, my conclusion is, that in healthy dairy cattle the temperature may vary between 100·4° F. and 102·8° F., with an average mean temperatue of 101·4° F.

Some Causes of Variations.

Time of Day.—Some of the causes and the extent of the variations of temperature in health are well worthy of note; and therefore I made 370 observations on a dairy of 37 apparently healthy cows, some in calf, and others not in calf. The temperatures were taken twice daily for five consecutive days: in the mornings, about 8 o'clock, and in the afternoons, between 4 and 5 o'clock, before feeding. The average morning temperature of 185 observations was 101·5° F., and the average evening temperature was 102° F. Thus we see an average rise of ·5° F. from morning to evening.

Feeding is usually credited with some slight influence in raising the temperature. In order to see to what extent this occurred, I took the temperatures of the same 37 cows during feeding between 4.30 and 5 P.M. The average temperature at that time was found to be 102·3° F. As the average temperature of rest of the same animals at the same hour on other days was found to be 102° F., it is permissible to assume that feeding was responsible for an average rise of temperature of ·3° F.

Drinking, immediately after taking the temperature, is usually responsible for a slight reduction; but I have no records at hand to show to what extent.

Pregnancy.—To see the extent of the rise caused by this condition, I made 60 observations on 6 apparently healthy, in-calf cows. Their average was 102° F. On comparing that with 310 observations made on 31 non-pregnant cows kept in the same cow-shed at the same time, the average of the latter being 101·7° F., a rise of ·3° F. is shown, presumably due to their pregnant condition.

Various other conditions are said to cause variations of temperature in health, such as œstrum, rumination, active lactation, housing, and the time of year. Hadschopulo states that, in cold weather, the temperature per rectum is raised from ·2° to ·4° F. (Quoted by Friedberger and Fröhner).

All these observations were made on housed dairy cattle. I hope at some future date to be able to record the results of observations made on store cattle kept outside.

The Temperature of Tuberculous Dairy Cattle not Clinically Affected.

It has long been an accepted fact that the temperature is of little assistance to the clinical observer in making his diagnosis in cases of tuberculosis in cattle by ordinary means; and one is not infrequently astonished, on examining the viscera of cattle in prime condition slaughtered for food, to find tuberculous lesions when least expected. Friedberger and Fröhner[1] say, "The temperature of the body in tuberculosis may be normal, although we far more frequently find an irregular remittent or even intermittent fever up to 41° C. (105·8° F.) . . . Clinical diagnosis is very uncertain. No diagnostic sign may be present, especially during the first few months of the disease."

Since the introduction of tuberculin as a diagnostic agent, however, tuberculosis may be detected in the very earliest stages, and where there has been no reason to suspect the presence of the disease. Tuberculin has now been in use for nearly fifteen years, and during that period has had a very extensive trial and proved itself to be almost infallible.

The observations here recorded were made on dairy cows that were not suspected of tuberculosis, which disease was only revealed by submitting the animals to the tuberculin test.

My observations number 505, and were made on 74 reacting dairy cows out of 137 tested. The temperature of each of the cows was taken on several consecutive days, and at various times of the day, and they were tested immediately afterwards. Obviously the temperature of these animals during the test period must not be included, since the reaction consists of a rise of temperature. The average temperature shown by the 505 observations is 101·7° F. (38·7° C.), which is ·3° F. higher than the average temperature of health. The lowest observed temperature was 100·4° F., and the highest was 104·3° F. The lowest average temperature of an individual was 100·6° F., and the highest average was 103·3° F. The greatest range of any individual observed was from 100·7° F. to 104·3° F.; while the average of fifteen observations on that same cow was found to be 102·2° F. Of the 74 cows in question, 50 per cent. had an average temperature falling between 101° F. and 102° F., while 10 averaged below 101° F., and 27 averaged 102° F. or over.

Thus, I am justified in concluding that tuberculosis in early cases causes only a slight rise in the average temperature, viz., ·3° F. but that affected animals are subject to greater variations from day to day than non-tuberculous cows kept under similar conditions.

A rather startling side-issue is revealed by the testing of these 137 cows, and that is the enormously large proportion of apparently healthy animals that reacted, viz., 74 out of 137, or 54 per cent.

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[1] "Veterinary Pathology:" translated by Hayes.

SUPPLEMENTARY NOTE ON ADENO-CARCINOMA OF THE LIVER.

By A. M. TROTTER, M.R.C.V.S., Glasgow.

IN a previous number of this Journal (Vol. XVII., p. 129), I described an adeno-carcinoma of the liver.

That article was penned after I had made observations on several hundred cases, and although the serous membranes were in every instance carefully examined no evidence of metastatic lesions was detected.

Since then one such case has come under my notice.

The subject was an aged Irish cow, in fair condition, and had been consigned from Limerick to Glasgow.

The primary lesion occupied the greater part of the left lobe of the liver. Its macroscopic characters were similar to those already described.

The substance of the liver was packed with secondary lesions which varied in size from a millet seed to a walnut.

A thrombus was present in the lumen of several branches of the portal and hepatic veins.

Members of the hepatic, renal, and right internal iliac groups of lymphatic glands were invaded. Each gland showed in its substance a metastatic lesion which had, in a greater or less degree, displaced the glandular tissue.

The parietal and visceral serous membrane of the peritoneal cavity was studded with nodules, which varied in size from a pin-head to a vetch. These nodules were more numerous in the anterior third of the peritoneum. They were firm and rotund. Their surface was smooth, and they had the appearance of being encapsuled. The majority were of a greenish colour, but some showed a more or less distinct yellowish tint. They were attached to the underlying membrane by bands of connective tissue. In some instances the bond was intimate, whilst in others a pedicle had been formed. The nodules, in places, had coalesced, and had formed more or less irregular areas. These areas were flattened and rarely exceeded in thickness 3 mm. Their margin, which was sharply defined and irregular, frequently overhung their attachment. Their surface was intersected by sulci formed by the junction of adjoining nodules. The sulci in places had become more or less obliterated through the formation of fibrous tissue, the fibrous elements being interwoven among the nodules.

On section, these nodules were seen to be formed of a homogeneous, somewhat translucent, material of a greenish colour. Some nodules showed evidence of having undergone degenerative changes. The areas, many of the component parts of which had undergone retrograde changes, were seen to retain in a great measure a nodular formation; in other words, the outline of the individual nodules was, with very few exceptions, discernible.

The microscopical examination of several of the nodules attached to the peritoneum showed that many were sub-divided into lobules, and that they were surrounded by fibrous tissue. This fibrous capsule was continuous with the sub-serous layer. The endothelium was, at places, seen to line the external surface. Numerous blood vessels were supported in the fibrous capsule. The fibrillar elements of the capsule were denser at the junction of the neoplastic tissue, from which it was separated by a well-marked line of demarcation. The outer layer of neoplastic cells were flattened through pressure. The cells were similar in appearance to liver cells. They were either round, oval, or irregular. They were supported by a fine fibrous net-work. The walls of the blood vessels were, as a rule, composed of a single layer of endothelium. The degenerations which these cells were more frequently seen to have undergone were chromatin and fatty.

The histological structure of the primary lesion was similar to that of the nodules adherent to the peritoneum.

TRYPANOSOMIASIS IN CATTLE OF INDIA.

By E. E. MARTIN, M.R.C.V.S., D.V.H., Capt. A.V.D.

IN an article appearing in the *Journal of Comparative Pathology and Therapeutics* of September 1904, by Durrant and Holmes, entitled "A Trypanosoma found in Blood of Cattle in India," some statements are made which are not correct, and liable to be very misleading. In the following number of the same Journal, in an article entitled "Some Diseases complicating Rinderpest among Cattle in India," Captain Holmes seems to have made an attempt to modify some of these statements, but it appears to me that they require some further correction.

The first statement I wish to question is, "Up to the present it has not been demonstrated that the cattle of India suffer from trypanosomiasis." Now, as far back as 1898 I was shown trypanosomes in the blood of cattle by Major Raymond, I.C.V.D., who informed me at the time that he found that both cattle and buffaloes suffered from spontaneous trypanosomiasis, but that the symptoms were confined to increased temperature, anæmia, and debility. He has made a practice for some years of having the temperatures of all cattle in the wards of the hospital regularly taken, and those that show any rise have their blood examined, and in this way many cases of trypanosomiasis have been brought to light which otherwise would not have been noticed. His theory is that cattle play the same rôle in Bengal with regard to surra as big game do in Africa to the tsetse fly disease.

The statement that immediately follows the above, namely, "It is known that cattle experimentally inoculated with the trypanosoma evansi are not affected," is contradicted by the authors themselves, when they state that cattle so inoculated do become affected, and that months after inoculation their blood is infective to other animals.

Later on in the article comes the following astounding paragraph: "In the *Veterinary Record*, 30th August 1902, there is an extract from an article by Laveran and Nocard describing a very fatal outbreak of surra among the cattle of Mauritius. This outbreak was ascribed to surra introduced by cattle imported from India. It is very improbable that this outbreak was caused by the trypanosoma evansi, which has never been noticed in cattle of India except when experimentally inoculated." This challenge by Durrant and Holmes to such workers as Laveran and Nocard is refreshing, especially as the former is now generally recognised as the highest authority on trypanosomiasis. When it is seen, however, that their challenge rests principally on the last sentence, "The trypanosoma evansi has never been noticed in cattle of India except when experimentally inoculated," it will be evident that it must fall to the ground, for, as I have endeavoured to show, this is totally incorrect, the spontaneous occurrence of trypanosoma evansi in cattle having been well known for years.

There is one other statement in this article to which I wish to call attention, and that is the one referring to piroplasmosis. They say, "Until recently it was not known that the cattle of this country were infected with piroplasma. In 1902 Stockman demonstrated the pear-shaped bodies. . . . Since then further investigation has shown that a very large percentage of cattle are affected." Though they do not in so many words ascribe the discovery of the piroplasma in India to Stockman, I think the reader will gather that this is what is intended. This is not the case, and I feel sure that Mr Stockman would be the first to disclaim any credit for a discovery that did not belong to him. In 1898 Major Raymond showed me specimens of the parasite in the blood of cattle, and in the same year embodied his discovery in an official report. In 1900 he published a "Note on Bovine Malaria," dealing very fully with the disease both from clinical and bacteriological points of view. This note was printed by the Government of Bengal Press.

With regard to Holmes' observation that in two cases of trypanosomiasis in bulls he found "peculiar developing forms bearing a striking resemblance to the crescents of human tertian fever," I would point out that Dr Hunt in 1897 called attention to these forms which he found in bovine piroplasmosis. Klein, to whom he referred his specimens, gave it as his opinion that they represented a stage in the development of the pirosoma. Raymond also afterwards found them in the cattle of Bengal, and he is inclined to the opinion that they have nothing to do either with trypanosomiasis or piroplasmosis, but that they are really sarcosporidia.

THE TRANSMISSIBILITY OF TUBERCULOSIS OF THE MONKEY TO THE OX AND GOAT, WITH OBSERVATIONS REGARDING THE REACTIONS OF THE EXPERIMENTALLY INFECTED ANIMALS TO TUBERCULIN.

By A. E. METTAM, B.Sc., M.R.C.V.S., Principal of the Royal Veterinary College of Ireland.

SINCE the meeting of the British Congress on tuberculosis, held in London in 1901, many researches have been devised and carried out with the object of examining the truth of Koch's conclusion that "human tuberculosis differs from bovine, and cannot be transmitted to cattle." It will be remembered that this statement of Koch's caused a certain amount of consternation, not only in the lay mind, but also among those who spend all their lives in the quest of the cause of disease, for the latter were familiar with the results of experiments which were in direct opposition to Koch's statement, and, further, many were acquainted with cases of accidental inoculation of tuberculosis into man. The eminence of Koch in the bacteriological world, however, demanded that his statement should be examined, and that new experiments should be carried out to determine if his opinion was correct or not. Commissions were appointed, both in this country and in Germany, and numerous other workers in public and private laboratories began experiments bearing on the whole question. Up to the present the findings practically amount to this, that Koch's statement is not correct, since human tuberculosis can be transmitted to bovines, though the human virus varies as to its virulency. Moreover, the results of the enquiry of the German Commission favour the conclusion which cannot be proved by direct experiment, that the bovine virus is fatal to man.

Practically the work when complete, if Koch's hypothesis is disproved, will establish what some of us thoroughly believe, that the tubercle bacillus is fatal to all animals, that tuberculosis attacks all animals from man to fish, and that the organism found in any species is not peculiar to that species, but merely the organism which has adapted itself to its environment, and perhaps has lost, and also perhaps gained, certain peculiarities which the original tubercle bacilli did or did not possess. Just as a micro-organism can be modified in culture media outside the body, so it may be modified in passing through the bodies of animals of the most diverse and varied species.

Recently the body of a drill monkey (cynocephalus leucophæus), which had died in the gardens of the Royal Zoological Society, came to me for *post-mortem* examination, and as I found the case to be one of tubercle, and as the disease was evidently the result of cutaneous inoculation and the virus probably of human origin, I determined, after passage of the virus through the organism of the guinea-pig, to see if it was virulent for the ox and goat.

The monkey had only been a short time in the possession of the Society, and, unfortunately, I am unable to give any of its previous history, but the *post-mortem* record is very interesting.

A bubo was noticed in each groin, the lymphatic glands were suppurating, and the left testis was purulent. A scar adherent to the subjacent connective tissues at the root of the penis probably indicated the site of infection. In the body the disease was limited to the abdomen and pelvis. There were no signs of disease above the diaphragm, the thoracic viscera being quite sound; and, strange to say, the liver with the exception of the portal lymphatics, spleen, and kidneys did not show any naked-eye lesions. The omentum was beset with tubercles of various sizes, and in the right sublumbar region, retroperitoneal in position, was a large tuberculous abscess, while another was present behind the peritoneum, subphrenic in position on the left side. The retro-vesical pouch was practically obliterated by a large tuberculous abscess. A few tubercles were found in the mesentery, and one the size of a small Barcelona nut near the mesenteric insertion into the cæcum. There was no recent tuberculous peritonitis, nor had there been any extensive tuberculous irruption at any time into the peritoneal sac. The omentum was loosely adherent to the abdominal wall. Minute examination of the alimentary tract from stomach to anus revealed two small tubercles in the submucosa of the ileum and a depressed healed scar upon the surface of the mucous membrane of the cæcum. I could not satisfy myself that this last was of a tuberculous nature, though the presence of the scar upon the mucous membrane roused suspicion; still, the tissues round about and beneath the scar on microscopical examination gave no evidence of tuberculous infiltration. Everything pointed to the healed wound of the perineum as the port of entry of the virus.

Smears were made and examined for the tubercle bacillus, the preparations being taken from a bubo, the testis, and the subphrenic abscess. The organisms, which were not numerous, were indistinguishable from those found in the sputum of human beings suffering from phthisis pulmonalis.

An emulsion of the pus was made in sterile water, and injected subcutaneously into two guinea-pigs. The injections were made on 28th October 1904.

On 10th December one of the guinea-pigs died, that is, forty-three days after infection. Tuberculosis of the inguinal and lumbar lymphatic glands and of the spleen had developed. An emulsion of the lumbar glands was made and injected at once into the peritoneal cavity of a female goat about two years old. She had been previously healthy, and in my possession some months.

On 16th December the second guinea-pig died with similar lesions to the first (forty-nine days after injection). An emulsion of the spleen was made, and 1 cc. was inoculated directly into the right pleural sac of a bull, about two years old, and in a vigorous condition. The bull did not react to tuberculin, with which it was tested prior to the inoculation.

Both animals were kept under close observation, a daily record being taken of their temperature at 1 P.M., but otherwise they were treated in precisely the same way as before inoculation.

Goat.—Between 10th December 1904 and 18th January 1905 the temperature fluctuated between 99·4° F. on 25th December and 102·8° F. on 16th December; these being the extremes, and the difference 3·4° F. Between 28th December and 18th January the temperature was fairly constant. The animal appeared in good health all the time and fed well. It was decided to test on 19th January with tuberculin to ascertain if infection had occurred. Thirty minims of tuberculin were injected at 7.30 A.M., with the result shown below :—

Date.		*Hour.*	*Temperature.*
January	18	10 P.M.	100·8
„	19	7.30 A.M.	101
„	19	10.30 A.M.	102
„	19	12.30 P.M.	102·1
„	19	2.30 P.M.	105·2
„	19	4.30 P.M.	105·8
„	19	6.30 P.M.	105·3
„	19	8.30 P.M.	105·3
„	19	10.30 P.M.	105·9
„	20	12.30 A.M.	104·8
„	20	9 A.M.	104·5

The temperature reaction of the goat following the injection of the tuberculin indicated infection with tubercle.

Bull.—As already stated, the bull, previous to infection, had been tested with tuberculin and had not responded. After inoculation the temperature, as in the goat, was taken daily at 1 o'clock, and the record was fairly constant. When inoculated the temperature was 101·8°, and from this time to the time of the tuberculin test the temperature varied between 2°. On 18th December it was 101°, and on 3rd January 103°. From 14th January to the morning of 19th January the temperature fluctuated little. On 18th January the temperature was 101·4°, and at 7.30 A.M., 19th January, 60 minims of tuberculin were injected, with the result shown below :—

Date		*Hour*	*Temperature*
January	18	10 P.M.	101·4
„	19	7.30 A.M.	101·4
„	19	10.30 A.M.	101·4
„	19	12.30 P.M.	101·2
„	19	2.30 P.M.	104
„	19	4.30 P.M.	105·9
„	19	6.30 P.M.	105·5
„	19	8.30 P.M.	105·3
„	19	10.30 P.M.	103·5
„	20	12.30 P.M.	103·3
„	20	9 A.M.	101·8

The reaction (4·6°) of the bull is typical of a tuberculous animal reacting to tuberculin.

The use of tuberculin as a test for tuberculosis which is not clinically evident is of great value. Though occasionally the unexpected may happen, still we may claim that there is no known method of revealing latent or minute lesions of tuberculosis so good or so reliable as the use of tuberculin. Its value is almost universally acknowledged, and its employment has got into the hands of laymen, who not only use it to test animals to discover if they are suffering from tuberculosis, but in some cases even to hide the very disease it is usually employed to reveal. Ever since it was first recognised as a diagnostic agent it has been held that a first dose of tuberculin gives immunity to a second dose injected within a month; in other words, that if an animal has received a dose of tuberculin it is useless to again subject it to a further tuberculin test until a month has elapsed. Manifestly if this is true plenty of latitude is given to those who wish to dispose of tuberculous animals and who know they will be submitted to the tuberculin test. It is mentioned that of some 41,808 animals going to Germany from other countries, and which were tested with tuberculin on the frontier and successfully passed the ordeal, some 7194 were found to be tuberculous when slaughtered. These animals had not reacted to the German testing, and it is presumed that they had been "tuberculinised" some short time before being presented on the frontier. It was suggested that a stronger tuberculin might overcome this temporary immunity and thus reveal the diseased; but it was pointed out that if it were known that a stronger tuberculin was being used then the first injection would also be made with a stronger tuberculin, and the second injection would still be of no effect. Professor Vallée, the successor of the lamented Nocard at the Alfort Veterinary College, has recently shown[1] that if a larger dose than usual is employed, and that if the temperature is taken every second hour from the time of injection, then it will be found that in tuberculous animals a reaction occurs earlier than was previously believed, and this even if the animal has had a dose of tuberculin only a short time before. The duration of the reaction is shorter than after the first injection. The increased dose which Vallée used does no harm to either diseased or healthy; they withstand the increased dose well. I determined to see the effect of an increased dose of tuberculin upon the goat and bull, and consequently they were both injected with tuberculin. The quantity was one and a half times that used on the first occasion.

Goat.—On 23rd January the morning temperature of the goat was 101·4°, the evening temperature 103°. The tuberculin was injected on 24th January at 7 A.M., with the following results:—

Hour.	*Temperature.*
7 A.M.	101
9.30 A.M.	101·9
11.30 A.M.	102·8
12.30 P.M.	103·9
1.30 P.M.	104·5
2.30 P.M.	105·3
3.30 P.M.	104·8
4.30 P.M.	106
5.30 P.M.	104·8
7.30 P.M.	104·5
9.30 P.M.	105
11.30 P.M.	104·5
1.30 A.M.	102·4
9 A.M.	102

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[1] "Revue Generale de Médecine Véterinaire," Tome iv., No. 40, 15th Aug. 1904.

The reaction is unmistakable. It is interesting to note that the highest temperature here, as on the first injection, occurred nine and a half hours after the injection. The rise in temperature on the second occasion was more gradual than on the first occasion, where the increase in height occurred between the fifth and seventh hours, and the maxima were attained at the ninth and fifteenth hours. The experiment shows that an increased dose of tuberculin will reveal tuberculosis even when injected a few days after the primary dose.

Bull.—The morning temperature on the 23rd was 101·3°, the evening temperature 99° (an unusually low temperature). At 7 A.M. on the 24th, when the tuberculin was injected, the thermometer registered 100·6°. The result is shown below:—

Hour.	*Temperature.*
7.30 A.M.	100·6
9.30 A.M.	102
11.30 A.M.	101·7
12.30 P.M.	102·6
1.30 P.M.	103·2
2.30 P.M.	103·2
3.30 P.M.	103·2
4.30 P.M.	103·3
5.30 P.M.	·103·7
7.30 P.M.	103·5
9.30 P.M.	103
11.30 P.M.	103
1.30 A.M.	102
9 A.M.	101·7

The rise in temperature (3·1° F.) in the bull is not so marked as in the goat, and one might hesitate to affirm that a reaction had occurred. But we must consider the temperature curve which extends over some few days to learn the bull's normal temperature under his present circumstances. From 13th January to 19th January midday the average temperature was 101·4°. If we take 101° as the average temperature, then the elevation was only 2·7°, and that occurred ten hours and a half after the injection. Probably the dose was too small, but if the case occurred in ordinary practice we should be justified in considering it suspicious and desirable to retest. This I resolved to do, and to give the animal this time a double dose of tuberculin.

The temperature was taken twice daily on 1st and 2nd February, and on the 3rd February, at 7.30 A.M., the animal received 120 minims, or about 7 cc. of tuberculin. The temperatures were as follows:—

Date.	*Hour.*	*Temperature.*
February 1	10 A.M.	102·2
„ 1	5 P.M.	101·8
„ 2	10 A.M.	101·8
„ 2	5 P.M.	101·6
„ 3	7.30 A.M.	101·3
„ 3	8.30 A.M.	101·4
„ 3	9.30 A.M.	101·7
„ 3	10.30 A.M.	101·4
„ 3	11.30 A.M.	101·3
„ 3	12.30 P.M.	101·3
„ 3	1.30 P.M.	102·2
„ 3	2.30 P.M.	103·2
„ 3	3.30 P.M.	103·5
„ 3	4.30 P.M.	103·5
„ 3	5.30 P.M.	104·7
„ 3	6.30 P.M.	104·3
„ 3	7.30 P.M.	103·8
„ 3	8.30 P.M.	103·8
„ 3	9.30 P·M.	103·2
„ 3	10.30 P.M.	103·0
„ 3	11.30 P.M.	102·9
„ 3	12.30 P.M.	102·4
„ 4	9 A.M.	102·5
„ 4	10 A.M.	102·3

In this experiment we may claim the result as positive, and it bears out the view that a reaction occurs to the injection of an increased dose of tuberculin, even if the animal has received tuberculin a short time before. The ten days which had elapsed allowed for elimination of much of the previous tuberculin and consequently for a loss of immunity. In this experiment the maximum temperature was registered at the tenth hour, and from this time it gradually fell to normal.

This single experiment shows that there is truth in Vallée's statement that a characteristic reaction will occur to a massive dose, but in our hands the reaction, as the careful hourly observations show, does not occur earlier than under ordinary primary tests. It shows, further, that more regular and constant observations are required after the application of the tuberculin or the result of the injection may be lost. It is generally understood that the reaction occurs at the tenth to the fifteeenth hours, and most often observations are made with the thermometer at the tenth, fifteenth, and eighteenth hours after making the injection. The charts show that approximately the tenth hour is a critical time, but it would probably be more satisfactory if the temperature were taken at short intervals from the seventh or eighth hour following the injection.

A very interesting reaction occurred in the bull after injection of this massive dose of tuberculin. At the time of inoculation there was noticed an escape of the virulent emulsion from the needle of the syringe, and some doubtless found its way into the subcutaneous tissues overlying the ribs. No reaction had, however, been noticed in the tissues, and the wound made by the needle had entirely distogether. We find that a marked reaction was obtained after the first injection; that a doubtful or equivocal reaction occurred at the second testing, probably because the dose was too small for the interval between the tests; that a large dose, double the ordinary, given ten days later, was able to reveal tuberculosis, though in this case the temperature reaction fell short of the first test; that the fourth test entirely failed, because of the immunity given four days before by the massive dose; and, lastly, the fifth dose gave a reaction comparable to the second dose in that the reaction was not very marked, though sufficient, possibly indicating the disappearance of immunity.

On the 17th February, at 7 A.M., I also retested the goat, this time using a smaller dose of tuberculin, viz., 20 minims. Prior to the application of the test the temperature for two days ranged between 101° and 102·6°. The result of this test was as follows:—

Hour.	*Temperature.*
7 A.M.	102·7
9 A.M.	102·5
10 A.M.	102·4
11 A.M.	101·6
12 Noon	102·2
1 P.M.	101·6
2 P.M.	103·2
3 P.M.	103
4 P.M.	103·3
5 P.M.	103·8
6 P.M.	103·8
7 P.M.	103·6
8 P.M.	103·6
9.30 P.M.	104·1
10.30 P.M.	103·6
12 Midnight	103·5
9.30 A.M.	102·8

The interval between the second and third tests was twenty-four days, evidently quite sufficient for any immunity given by a large dose of tuberculin to disappear, and we find that a dose of tuberculin smaller than that given on the first occasion was capable of giving a reaction. The reaction, however, was not so vigorous as on the first occasion, and its appearance was longer delayed.

CONCLUSIONS.

Tuberculosis of the monkey can be transmitted to the ox and the goat.

Tuberculin if given in doses above the ordinary reveals tuberculosis even if the animal has already within a short period of time received the usual dose as a test.

Tuberculin does give a certain degree of immunity to tuberculin, but this immunity may be overcome by giving massive doses.

An animal which has received a massive dose of tuberculin will not react to an ordinary dose of tuberculin, though known to be tubercular and capable of responding to tuberculin.

In those cases where there is reason to suspect the prior use of tuberculin it is justifiable to use an increased dose at the time of testing. This increased dose will do no harm.

An immunity to tuberculin may have disappeared after a lapse of ten to fourteen days, and an ordinary dose of tuberculin will then cause a reaction in a tubercular animal though it has been tested previously.

The reaction is most likely to be pronounced if a massive dose is given.

A small dose of tuberculin will give a reaction, but the reaction will probably be delayed.

I desire to acknowledge here my great indebtedness to Mr A. C. Duncan, M.R.C.V.S., Assistant in the College, for the care he took to make the temperature records exact and trustworthy.

ON THE CO-RELATION OF VARIOUS DISEASES IN STOCK IN SOUTH AFRICA.

By A. THEILER, Veterinary Bacteriologist, and S. STOCKMAN, late Principal Veterinary Surgeon, Transvaal.

IN an article bearing the above title which was presented at the 1903 meeting of the South African Association for the Advancement of Science, and which appeared also in this *Journal*[1], Dr Edington makes certain statements of a very far reaching character. Fortunately, however, the experiments and observations from which the author draws his deductions are given in detail. We say fortunately, because were some of the author's most important statements correct, the position of stock raising in South Africa would be a most hopeless one, and the fabric of the pathology of some of the most important diseases which has been laboriously built up by many earnest workers would represent so much wasted energy and misdirected reasoning. As many of Dr Edington's portentous conclusions were in direct opposition to our own experimental findings and field observations we felt compelled to take exception to them ; but in order that there might be no misunderstanding due to experimentation which was not exactly parallel, we decided to repeat his experiments on a still larger scale than that undertaken by himself.

On account of the importance of the subject we propose to discuss the author's conclusions *seriatim*, and record certain experiments which appear to be in direct opposition to his results.

(I.) "That the diseases known as horse-sickness, heartwater, impunga, boschziekte, gall-sickness, veldt-sickness, blacklung, and riverziekte should be regarded as one, or phases of one, malady."

We admit that with the exception of horse-sickness, which is a disease of the equine species, the various names mentioned are given to diseases in cattle, but we deny that these diseases are always the same. Our experience has taught us that by these local names many and various diseases are designated, to which belong such well-known maladies as heartwater, redwater, and coast fever, as well as others which may be caused by vegetable poisoning. In a country where the nomenclature of disease has been at the mercy of laymen it was only to be expected that the same disease would have received different names in various parts, and that modifications of the same malady would be designated by others which give the idea of distinctness. It is, therefore, unwarrantable to identify these names with heartwater or horse-sickness.

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[1] "The Journal of Comparative Pathology and Therapeutics," Vol. XVII., Part 2, June, 1904.

(2.) "That heartwater can be transmitted to cattle."

We take no exception to this statement, and give Dr Edington the credit of having demonstrated this in the first instance.

(3.) "That horse-sickness can be transmitted to cattle."

Seeing that horse-sickness is a disease which is directly inoculable, nothing should be easier than to prove this experimentally. We are in a position to record some experiments bearing on the point.

Experiment I.

Ox	No. 1	injected with	100 cc.	horse-sickness	virus.
„	No. 2	„	100 cc.	„	„
„	No. 3	„	200 cc.	„	„
„	No. 4	„	200 cc.	„	„
„	No. 5	„	650 cc.	„	„

None of these animals showed any reaction, notwithstanding the large amount of virus inoculated. The five oxen were bled on the ninth day after inoculation, that is to say, at a time when horses so treated would show a typical reaction of horse-sickness.

The blood was injected into five horses, Nos. 99, 102, 103, and 104 in quantities of 100 cc., and 200 cc. in horse No. 100. The injection of the ox blood into the horses had an immediate effect, which was to be expected, and is explained by the toxic effect of the ox serum. The horses showed uneasiness, slight colic, and hurried respiration. Horse 100 (which received a double dose) was very sick, and suffered from colic in a severe form. He purged slightly and strained in passing urine. The result, however, was negative, as none of the horses showed any reaction indicative of horse-sickness. The horses were afterwards used for other experiments, and died of horse-sickness.

Experiment II.

Calf 239, born in the Pretoria Station, was injected subcutaneously with 10 cc. of blood from Horse 658 while suffering from horse-sickness which proved fatal.

The following animals also received 10 cc. of the same blood:—

Ox	245	born in	Capetown.
„	249	„	„
Heifer	279	„	Aliwal North.
„	274	„	„ „
„	260	„	Capetown.
„	263	„	„
„	257	„	„
„	254	„	„
„	277	„	Aliwal North.
Calf	240	„	Pretoria Station.
„	241	„	„ „
„	242	„	„ „
„	243	„	„ „

The animals used in this experiment were, as shown above, collected from three different quarters—from Pretoria, from Capetown, and from Aliwal North. The Pretoria calves, which were stable born, had passed the last horse-sickness season in stabulation. Capetown and Aliwal North are parts of South Africa where horse-sickness is not known; it could not be objected, then, that the animals had acquired immunity from a former attack. Nevertheless, none of these animals contracted horse-sickness or showed any symptoms due to that disease. We are, therefore, we think, entitled to state that horse-sickness cannot be inoculated to cattle. We have additional reasons for refusing to accept Dr Edington's statement; to these we shall allude later.

(4.) "That horse-sickness can be transmitted to goats."

The following experiments were undertaken:—

Experiment I.

Goat (no number), born on the premises (Pretoria), was injected with 10 cc. of horse-sickness virus.
Goat 211, Angora, imported from the Cape „ „
„ 209, „ „ „ „ „
„ 208, „ „ „ „ „
„ 207, „ „ „ „ „
„ 206, „ „ „ „ „
„ 205, „ „ „ „ „
„ 204, „ „ „ „ „
„ 139, „ „ „ „ „
„ 133, „ „ „ „ „

None of these animals took horse-sickness. We may, therefore, conclude that horse-sickness is not inoculable to goats.

We admit that in the above experiments we purposely did not attempt to obtain preliminary evidence that the bovines and goats inoculated with horse-sickness virus were not proof against heartwater. We were convinced that if we attempted this by inoculating heartwater virus our animals, owing to death, would probably not be available for experiment with the horse-sickness virus. It may be stated, however, that out of the same lot of cattle and goats, which were purposely imported from the Cape (a clean district) for heartwater experiments, several animals were afterwards experimentally infected with heartwater and died of the disease. In fact, none of this lot which received heartwater virus proved refractory. This indicates that the above cattle and sheep which were not born in heartwater districts were susceptible to heartwater.

Our other reasons for repudiating Dr Edington's conclusions are based on important considerations of locality. As far as we are aware, Dr Edington was working in an area where both diseases, horse-sickness and heartwater, are observed, and we are inclined to think that this may in a measure be responsible for the conclusions which he has drawn. In Pretoria, however, we were working in an area where only horse-sickness is met with, and no heartwater is observed. Now, we know that heartwater is transmitted under natural conditions by a certain species of tick (amblyomma hebræum), and in this way only. This species of tick does not exist in Pretoria, nor is it found in any part of the Transvaal where the bush is absent, but where horse-sickness may be rampant. Truly enough, wherever heartwater is found horse-sickness is also met with, but the converse does not obtain. These observations, which could certainly have been made in Cape Colony as well as in the Transvaal, are of themselves almost sufficient proof that the two diseases are distinct.

(5.) "That the spontaneously occurring cases of veldt-sickness present *post-mortem* phenomena which are more or less identical with those of cattle dying from horse-sickness or heartwater."

It is not surprising that the *post-mortem* lesions of veldt-sickness should resemble those of heartwater, seeing that the names are most frequently applied to the same disease, but when Dr Edington stated "horse-sickness and heartwater" it inclined one to the belief, even in the absence of refuting evidence of an experimental nature, that the disease he observed in cattle after the injection of horse-sickness blood was heartwater naturally acquired. That the *post-mortem* changes found in horse-sickness are somewhat similar to those met with in heartwater we admit, but that is not sufficient proof of their pathological identity.

(6.) "That cattle born in the Karoo are liable to die when transported to the coast districts, and this disease is known as impunga or veldt-sickness."

Cattle born in the Karoo are susceptible to heartwater, and also to redwater, being born in districts where these diseases do not exist. The same holds good for cattle born in the Karoo and brought to the Transvaal Bush veldt, but there we apply the names heartwater and redwater to the diseases from which they die. We cannot accept that it follows because the name impunga is given to a disease which is probably heartwater in cattle, and this disease is similar in *post-mortem* symptoms to horse-sickness, that these cattle going to the coast from the Karoo die of horse-sickness.

(7.) "That heartwater may be transmitted in a non-virulent form to goats, etc., and by transmission of blood from goat to goat in a series, can be ultimately raised in virulence."

We doubt seriously that part of the statement which refers to the exaltation of virulence, and assumes the possibility of obtaining a "virus fixe" of previously unsuspected virulence. The point is important, because it is claimed, apparently, that Horse A was successfully inoculated with horse-sickness by means of heartwater blood owing to the abnormally exalted virulence of the latter.

We think there is another very probable explanation of the apparent exaltation of virulence. There are goats born in heartwater countries, and those which, although not so born, have been introduced, and in both these classes there are many animals which are highly immune owing to a former attack of the disease. Inoculation with blood in the latter produces only a passing reaction of little consequence, but one often finds amongst a batch of experimental animals certain individuals possessing no immunity at all. We freely admit that a great difficulty in the way of correctly interpreting the results of many experiments performed in South Africa lies in the animals available not being free from reproach.

(8.) "That heartwater blood cannot be preserved in a virulent form, while, conversely, horse-sickness blood can be so preserved for an indefinite period."

This statement is undoubtedly right, but we think we are justified in using it as evidence against the author's conclusion that the two diseases are the same. We do not know that such an analogy exists in any known disease which is inoculable into various species of animals.

(9.) "That the blood of cattle dying of veldt-sickness can produce, by forced infection and subsequent transmission from goat to goat, typical heartwater."

Veldt-sickness in cattle is identical with heartwater. Therefore, it was to be expected that heartwater would result in susceptible animals from inoculation. Heartwater, however, is admittedly a disease of cattle, sheep, and goats, and it is inoculable from susceptible individuals of one species into those of the other without forced infection, but this cannot be fairly held to support conclusion 1, in so far as it applies to horse-sickness.

The author of the note continues, "The corollary to these experiments would have been a demonstration that heartwater could be conveyed to horses." "Unfortunately, all attempts even by the intravenous injection of huge doses of heartwater blood failed to produce anything more than a transient elevation of temperature in the horse." This might have led to the conclusion that the two diseases are distinct, but another reason is sought for to explain the failure, viz., that the virus employed was in some way attenuated, and he finally claims to have successfully conveyed horse-sickness to horses by using heartwater virus of exalted virulence. The evidence in support of this important statement merits analysis. He exalts his virus, as before mentioned, by passing it through clean goats, and he finally injects two horses, A and B, with 20 cc. of blood from a goat dying of heartwater. The temperature of Horse A rose on the sixth day, and he died on the morning of the twelfth day. The animal shows on *post-mortem* changes which are characteristic of horse-sickness, but, and this is important, in addition to the ordinary changes there was a yellowish discoloration of the serous exudates, which is somewhat more commonly associated with the disease known as biliary fever. Goats injected with the same virus for control purposes died of heartwater, but there is good reason to question whether Horse A died of horse-sickness. Indeed, from the author's own statement, one must strongly suspect that the horse died of biliary fever. We may here mention that it is not uncommon to find a purplish colour of the stomach in the latter disease, and the two maladies are not infrequently mistaken for each other by quite experienced persons. The subsequent inoculation of this horse's blood into another horse points strongly to biliary fever, for again particular stress is laid on the yellowish discoloration of the exudates, which is unusual in horse-sickness.

Knowing well the difficulty of experimenting with such diseases in an infected area, we are inclined to think that Horse A contracted biliary fever accidentally, and that it was afterwards transmitted by inoculation. With regard to Horse B, which did not die after inoculation of ½ cc. of preserved horse-sickness blood, and according to the author's conclusion must have been salted by the heartwater virus, we feel bound to point out that in using horses picked up at random for horse-sickness experiments, one meets unexpectedly with individuals which have naturally acquired immunity, and everyone who has lived in South Africa knows how this has occasionally led to patent preventives acquiring considerable repute.

This single horse, then, which resisted horse-sickness virus may very likely have been a naturally salted one. The experiments which we are about to quote support this interpretation.

According to Dr Edington's statement, a very exalted virus of heartwater, obtained from serial passages through goats, is able to produce horse-sickness in horses. We found ourselves in a position to repeat these experiments. Since 1903 a series of heartwater experiments have been in progress at the Pretoria station. The strain of virus was obtained from the Cape colonial entomologist, Mr Lounsbury, who kindly sent the infected ticks. This statement is important, because Dr Edington drew his virus from the same source. The virus was kept up by inoculation from goat to goat. After many passages we may conclude that our virus is similar to that used by Dr Edington. An Argentine horse and an Argentine mule were inoculated with this virus. These animals were freshly imported. They came directly from the ship by train to our premises, which they never left. Everything was, therefore, in favour of their not being immune to the disease of the country.

On 29th August 1904 horse 606 was injected with 20 cc. of heartwater blood. No reaction took place.

On 20th October 1904, the horse was submitted to the immunisiug process against horse-sickness, which entails inoculation with virulent blood. A typical reaction set in, and the horse passed through horse-sickness. On 29th November 1904, the horse was injected with blood from a case of biliary fever. He contracted biliary fever and died on the 13th December.

Mule 594 was injected on 29th August 1904, with 20 cc. of heartwater blood. No reaction ensued.

On 30th December 1904, the animal was used for the horse-sickness experiment in which the blood of a sick country-bred animal was used as virus for the purpose of noting the influence of the two diseases, horse-sickness and biliary fever, which are sometimes transmitted by using such blood. The mule went through a horse-sickness reaction, but the temperature did not drop at the typical time, and, as was anticipated, biliary fever developed. On the twelfth day after inoculation red urine was voided, and the piroplasma was very frequent in the blood. The animal recovered from both diseases. These two experiments are not open to the same reproach as those done in Cape Colony, and in all likelihood the same strain of heartwater virus was used. That the animals were not immune against horse-sickness was proved by the subsequent inoculation, which gave a typical reaction of horse-sickness.

CONCLUSION.

1. The same diseases of stock may have many names in South Africa.

2. Different diseases may have the same name.

3. The disease in horses known as horse-sickness resembles in *post-mortem* lesions a disease in cattle, sheep, and goats, known as heartwater. These diseases cannot be identical, since they are not inoculable respectively from horses to ruminants or *vice versa.*

4. Heartwater is inoculable to all domesticated ruminants. Horse-sickness is only inoculable to equines.

CANCER OF THE HORN-CORE OF CATTLE.

By K. HEWLETT, M.R.C.V.S., Indian Civil Veterinary Department.

THE disease known as "Horn-Core Disease of Cattle," or "Cancer of the Horn-Core," is met with not very infrequently among cattle in the city of Bombay Presidency.

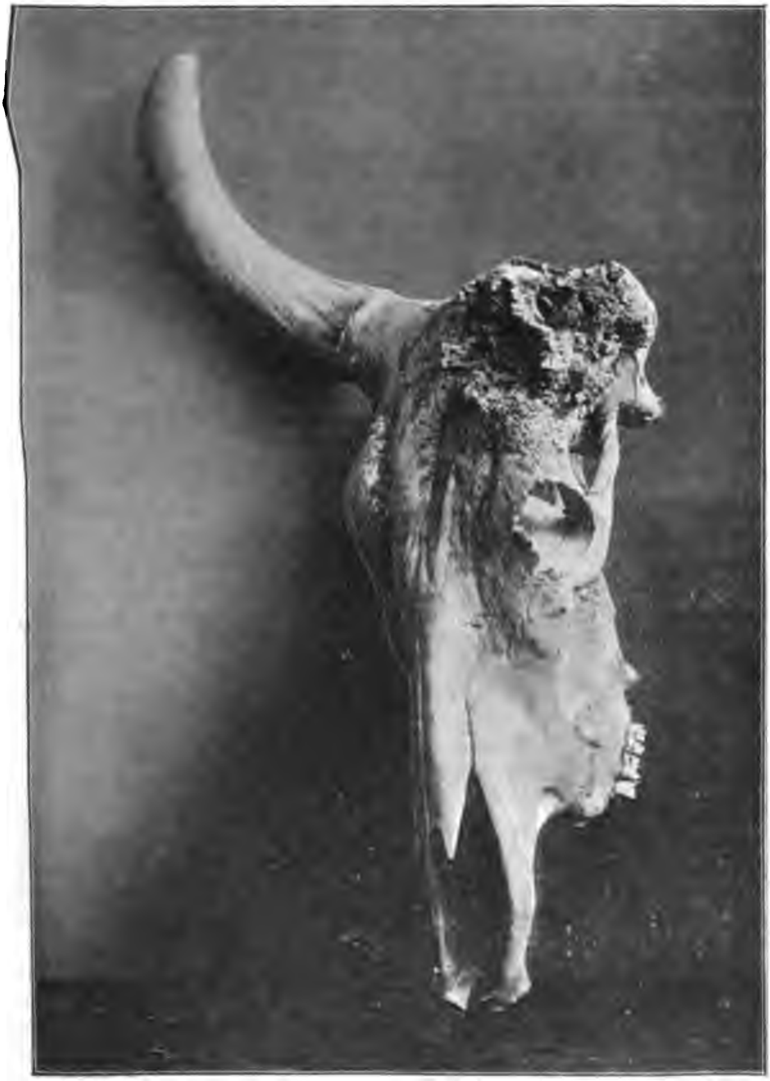

FIG. 1.
Skull from a case of cancer of the horn-core.

I have during the last two years attended twelve cases of this disease in the hospital attached to the Bombay Veterinary College.[1]

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[1] This, however, is not to be taken as a measure of its frequency. On the contrary, the majority of natives do not seek advice for their animals from the European veterinary surgeon.

The disease obtains its name from the fact that the horn-core is the part most prominently affected.

Various breeds of cattle suffer from the disease, and all the cases which have come under my observation were aged working bullocks.

In most cases no history has been available, but in one the owner stated that the animal had received a blow on the horn some time previously.

In the majority (about two-thirds) the left horn-core is attacked. I have not observed or heard of a case in which both horns were affected.

A slight drooping of the horn is the first symptom noticed. The drooping increases until the horn hangs down and becomes loose. There is then noticed a distinct line of separation between the horn and the skin. Sometimes a greyish, thick, fœtid material exudes from this cleft. Later the loosening of the horn increases, and by moderate traction it may then be removed, leaving the altered horn-core exposed. This presents a greyish moist appearance. It is often very fœtid, and in places quite pulpy. Its consistence in other parts, however, is firm, but it is easily cut with a knife. The bony structure of the horn core is completely lost, and there is no evidence of its normal cavity. Towards the base the growth is sometimes very vascular. An ostitis often affects the frontal bone at the base of the horn core (Fig. 1). Sometimes after amputation the frontal sinus is seen to be filled with the new growth.

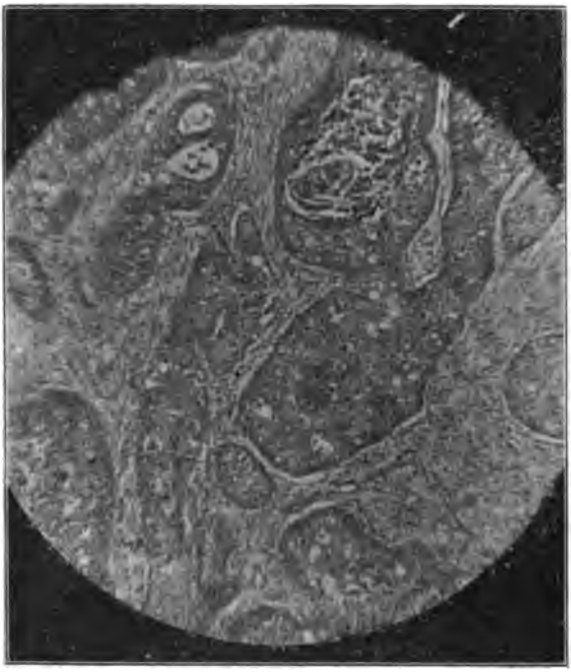

FIG. 2.

Micro-photograph (× 105) of growth from a case of cancer of the horn-core.

On several occasions I have attempted amputation of the horn and extirpation of the growth, but each time the latter rapidly recurred. Most of the cases have come under my observation after the horn had been amputated about two inches above its base, and then the growth presented a cauliflower appearance.

The disease does not appear to cause the animal much pain, and as a rule there is no constitutional disturbance; but as the growth progresses there is considerable loss of condition and wasting.

I have been unable to make *post-mortem* examinations on these cases on account of the religious prejudices of the owners.

When Mr Stewart Stockman, late of the I.C.V.S., was in Bombay, I showed him several cases which were then under my care at the Veterinary College.

He expressed the opinion that the condition was probably due to a malignant growth, of which he undertook to furnish me with a histological description if I would collect clinical notes on such cases as came under my notice. From a microscopical examination of tissue from six cases Mr Stockman informs me that the disease is an epithelioma, which probably begins in the mucous membrane of the sinus, from which it invades the horn core, and excites ostitis in the bones.

The accompanying microphotograph (Fig. 2), which is supplied by Mr Stockman, shows the histology of the new growth.

FURTHER EXPERIMENTS TO DETERMINE HOW LONG AN AREA REMAINS INFECTED WITH EAST COAST FEVER.

By A. THEILER, Government Veterinary Bacteriologist,
and S. STOCKMAN, late Principal Veterinary Surgeon, Transvaal.

I.—Purification of Infected Ground.

IN a former article on the above subject certain experiments were described showing that cattle could be introduced with impunity on a pasture which about fourteen months previously was badly infected with East Coast fever.[1] We decided to continue these experiments on other farms and under varying conditions. At the same time we drew up a form of enquiry to be answered by farmers who had had East Coast fever on their farms, in order to ascertain their experiences on the subject, and compare them with the result of our own experiments.

The information obtained was rather meagre, as the returned forms represented but a small percentage of those issued.

There were only a small number of the observations which corresponded exactly with the conditions under which our experiments were started, and in interpreting the results we were met by new factors, the influence of which it was difficult to judge accurately.

It will be remembered that on 15th December 1903 ten Texas heifers were exposed at Mr Hall's farm, Nelspruit; that on this farm for a period of fourteen months no other cattle had been running; and, that it had been badly infected prior to that time. In fact, a large herd of cattle thereon had been wiped out. The outcome of our re-stocking experiment was that none of these heifers contracted East Coast fever, although they continued on the identical grazing ground

[1] Theiler and Stockman: "Journal of Comparative Pathology and Therapeutics," Sept. 1904.

up to 30th May 1904 (four-and-a-half months). On this date the animals were removed to another place which had also been previously infected. Mr Hall, encouraged by the result of our experiments, decided to bring eight Madagascar oxen to the farm on which our heifers had been grazing. This he did, after obtaining the necessary permission to move cattle into an infected area and the assistance of the railway authorities to detrain the oxen directly on to his farm, through which the line passed. These oxen have since been on what was at one time very badly infected ground, yet none of them have contracted East Coast fever. This observation may be looked upon as a continuation of the re-stocking experiment started on the farm by us, and which has now lasted about eighteen months.

The Texas heifers which were running up to the 30th May on Mr Hall's farm were, as already mentioned, placed on a new piece of fenced-in ground. On this actual spot twenty-eight head of our experimental cattle had died during January and February of 1903, all within an area little larger than 3 acres. The *post-mortem* records and microscopical examinations of the blood of these twenty-eight animals proved beyond doubt that they died of East Coast fever. No cattle had since been on the ground, and in the month of November 1903 (*i.e.*, nine months after the last animal died) it was fenced off, in order to prevent any possible re-infection by cattle straying on to it. As a matter of fact, there were then no cattle which could stray on to the ground, but we desired to surround ourselves with the conditions demanded for exact experiment.

After the erection of the fence, fifteen sheep and goats were placed in the paddock. These, with the exception of four, died from heartwater. The surviving four were returned to Pretoria on 13th April 1904. Our experimental Texas cattle were put into this paddock on the 30th May 1904, almost exactly fourteen months after the last animal had died. To get there, they were driven over a stretch of ground which about fifteen months previously had probably also been infected with East Coast fever. From the 30th May 1904 up to 31st March 1905, one death has taken place, namely, during November 1904. This was not the result of East Coast fever, as the *post-mortem* and the microscopical examinations gave negative results. These cattle then, had grazed for fully nine months on ground which fourteen months previously had been badly infected with East Coast fever, and on which the brown tick was prevalent.

The result of this experiment supports that of our former observation made on Mr Hall's farm. There is this difference, however, in the conditions, namely, that in the present case the pasture had been grazed over by fifteen sheep and goats. These animals, which are immune to East Coast fever, might have picked up the infected ticks, and the latter lose their virulence after biting on a clean or immune animal. Engorged ticks were collected from the sheep for breeding purposes. The most common varieties were the following, given in the order of their frequency: hyalomma ægyptium, amblyomma hebræum, rhipicephalus simus, and rhipicephalus evertsi. It is noteworthy that few brown ticks were collected from the sheep and goats, although they had been found in great numbers on the cattle which had died there. This fact, however, does not exclude the possibility of a larger number of brown ticks having been picked up by the sheep and goats, because the brown tick does not stay long on a host, but we think the number cannot have been very great, otherwise they would have been noticed more frequently with careful observation. We may, therefore, conclude that the purification of this piece of ground was not so much due to the agency of the small flock of sheep and goats as to the lapse of time which passed between the death of the sick animals and the introduction of the green cattle.

Another experiment, which by now may be considered concluded, was undertaken in the neighbourhood of Barberton. This was started, at our instigation, under the auspices of the Agricultural Society of Barberton, some members of which were very anxious to test for themselves the practicability of purifying infected areas of that district with a view to re-stocking.

A meeting of the Society was held on 10th September 1904, at which we were invited to attend and explain the course to follow, and a sub-committee was formed to advise us as to most suitable previously infected areas for purposes of demonstration. A piece of ground was selected on Mr Niekerk's farm, and sufficient grazing was fenced in to carry eight oxen. Cattle had died of East Coast fever on this particular ground about fourteen months previously.

Some delay in putting up the fence unfortunately took place, and, in consequence, the fresh cattle, which had to be brought by rail from a clean district, could not be placed on the farm until 21st November 1904. Eight oxen were exposed in the first instance. On 1st December 1904 nineteen other oxen were temporarily placed on the same farm, but they had to be removed owing to the scarcity of grass. Our desire was to obtain a piece of ground which had been infected twelve months previously and had since been free from cattle, but, as we could not obtain this in Barberton at a reasonable price, the nineteen oxen were brought up on the 10th January 1905 to Nelspruit, where in the meantime one of our marked-out areas was ripe for the experiment. It must be explained, however, that owing to the cupidity of the farmer who was to receive certain oxen for his trouble, the original eight which were sent to his farm were not retained there. He kept eight out of the second lot of nineteen which were fatter. The experiment, then, was really not carried out as originally intended, but the subsequent results show that the substitution had no real influence on the result. Since no death due to East Coast fever occurred either amongst the cattle on Niekerk's farm, or later at Nelspruit amongst the lot sent, we may justly conclude that the piece of ground at Barberton which had been infected about fifteen months previously had become purified.

In our former article we described certain experiments at Nelspruit which were carried out during the months of November and December 1903, and January 1904. We must refer to these again in order to state that during their course fifteen animals died of East Coast fever in November 1903, eight died during December, and seven died in January. All these animals contracted the disease within a certain area known to be infected, and which we kept for various experimental purposes.

The date of the last death from East Coast fever amongst the experimental animals on this area at Nelspruit was 25th January 1904. Out of forty-six animals which we exposed on the ground, only one survived. The temperature of this surviving animal was constantly taken, and whenever any disturbance took place a microscopical examination was made of the blood, and this in every case gave negative results. We therefore concluded that this animal was one of the rare exceptions where an ox shows natural immunity to the disease. Since November 1903 five immune oxen, for shortness called "the Komati oxen," the survivors of a herd of 500 which had perished in August 1902 at Komati Poort, had been running on the same infected pasture as the above-mentioned cattle which had died. The five were taken to Pretoria on 17th January 1904 to be used for inoculation purposes—the preventive method of Koch, who advised that the blood of recovered oxen is protective. These immune animals, namely, the one survivor from our experiment and "the Komati oxen," were the only animals which grazed on the ground from 25th January to 13th April 1904.

On 13th April three fresh heifers were exposed for the purpose of obtaining infected ticks, namely, Nos. 200, 222, and 234. Heifer 200 was returned to Pretoria on 25th June 1904, being then in high fever. The cause of this febrile condition could not be ascertained, but East Coast fever can be excluded, since the heifer recovered, and at no time could the piroplasma be found in the blood. Heifer 222 died on 13th October from heartwater, and microscopical examination of the blood excluded any East Coast fever infection. Out of this lot of three animals there remains one alive on the Nelspruit Experimental Farm at the time of writing.

On 22nd June 1904 ten oxen were sent to the same place. Seven of these had been inoculated according to Professor Koch's method, and were then ready to be exposed for testing. Three had not been injected with Coast Fever blood; they were to serve as controls. Of this lot (controls) one ox died suddenly, namely, No. 239, on 10th January 1905. Unfortunately no smear was obtainable for microscopical examination, as the assistant in charge was absent on duty elsewhere at the time, but the record of temperature which was taken twice every day indicated clearly that the animal did not die of Coast fever. The temperature was normal up to the day of death, an unheard of thing in East Coast fever. The animal probably died of vegetable poisoning, a not uncommon occurrence.

On 8th December 1904 the five "Komati oxen" (naturally salted), which on 13th January 1904 were sent to Pretoria, were returned to Nelspruit, simply to get them out of the way, and there they remained.

On 10th January 1905 the nineteen oxen sent on 1st December 1904 to Barberton were brought to Nelspruit. At that time eleven and a half months had passed without a single case of Coast fever occurring on our ground. It was considered that if a larger number of oxen were exposed it would demonstrate more clearly whether or not the pasture had become purified within these eleven months. That is to say, in all likelihood a greater number of ticks, including infected ones, would be picked up, if the latter still existed. No case of Coast fever, however, was recorded. We may, then, conclude that the once badly infected area of our Nelspruit Experimental Station had become purified, and, what is particularly striking, it had become so during the period in which susceptible cattle and immune oxen were grazing upon it.

A glance at the record of blood examinations during the first few months of the year 1905 shows the presence of Coast fever in the various infected districts. Most of the outbreaks occurred in the month of February. This record may serve in a sense to control the experimental one just described, for it shows that the general conditions in the country had not changed in the same way as they had changed on the Nelspruit farm, which at one time was notoriously infected.

The change of affairs at Nelspruit is quite explicable, but, in order to understand it, it will be necessary to explain how the experiments were conducted. The temperature of every exposed animal was taken morning and evening, and as soon as a temperature reaction was observed the animal was removed to a stable, caps were drawn over the ears, and all engorged ticks eventually collected in these caps. No sick animal was allowed to run on the farm, and death eventually took place in the stable. It will be remembered that once a virulent tick has bitten on a clean animal its poison is discharged, and it will be evident that by removing the sick animals from the pasture other ticks could not infect themselves upon them, and continuous reinfection of the ground was prevented. By removal of the infected ticks, then, the disease had died out.

The experiment also proves that immune oxen are not infective, and do not continue to infect the pastures as was asserted by Professor Koch. Had Professor Koch's assertion been correct this pasture should have been permanently infected, as immune oxen were constantly running on the place. We think that the presence of the latter probably helped to purify the ground, as infected ticks (larvæ or nymphæ) which suck on non-susceptible animals lose their virulence. A question which arises out of our experiments is, whether the purification of the ground is due to the disappearance of the brown tick, or to its virulence being lost.

As already stated, a collection of all procurable ticks was made from the sick animals which were forwarded to Pretoria, and they served for further experiments which will be referred to later. The collection of ticks from the exposed susceptible and immune animals was continued, with the result that brown and black pitted ticks, principally the former, were constantly found on the immune cattle, as well as on the three non-immune which were exposed on 13th April 1904, and did not take East Coast fever. There was, however, a distinct decrease in the numbers during the month of April, and in May, June, and July none were collected. The absence of brown ticks during these months can be explained by the approach of the cold season, and by burning of the grass which then took place. The burning of the grass is an annual function, which was carried out against orders. It happened at a time when only a few cattle were on the ground.

In August the ticks became more numerous again (principally nymphæ were collected both from the immune and the non-immune cattle). The same thing continued during September and October, and in November the adult ticks again appeared. They were, however, found in small numbers. In December none were met with, and only one specimen was obtained in January. After that no more were found, although other ticks such as bont, bont-legs, and red-legs ticks were constantly found. We were face to face, then, with the fact that the brown tick had almost completely vanished in an area which was once badly infected. The disappearance of the disease, however, did not synchronise with the disappearance of the tick; brown nymphæ and adults were found on all the susceptible animals at a time when infection could reasonably by expected to take place, namely, within six months after the death of the last animal on 25th January 1904.[1]

That the place did thereon not become reinfected was undoubtedly due to the fact that we did not allow any sick animals to graze. The infected ticks which missed being picked up and taken away by the cattle in the earlier days were either dead before the other susceptible animals arrived, or they had lost their virulence. In addition to the immune oxen other non-susceptible animals were running on the ground—four camels and some mules. We also found the brown tick on a wild hare. All these animals, we consider, took part in purifying the ground.

The conclusion to be drawn from these observations is of the greatest practical importance, viz., that it is possible to cleanse an infected area even when there are susceptible cattle running on it, provided the cattle are removed as soon as they become sick. Use can be made of this observation in practice to save cattle when the disease has broken out in a herd. All cattle should then be confined in a small non-infected area, and the temperature of every animal should be taken for twenty days. This will enable one to divide the herd into two lots—the sick, and those not yet affected. The former are either killed or returned to the infected ground, and the latter remain on the clean pastures. This method of purification is slower, less certain, and in some cases more expensive than if all the cattle, suspected and affected, are removed for slaughter. Moreover, on small and badly infected farms the clean ground available is insufficient, but it is more in accordance with the prejudices of the farmers, and on big farms it is quite feasible.

Now, we know that the incubation time of Coast fever at its longest is twenty days. Therefore if we take the temperature at intervals during this time and act in the manner above indicated, there is no danger during the period of non-infected cattle becoming infected by ticks which may have dropped from their sick fellows on clean ground, because the ticks which drop to the ground must, in the first instance, undergo a moulting process before they are able to reinfect fresh cattle, and this period lasts more than twenty days; nor is there a very great danger of the new ground being badly contaminated by sick animals removed in the incubation stages provided they be killed or removed as soon as the temperature rises, because the ticks do not take up the infection from the blood during the incubative stages of the disease. After twenty days all the oxen which have shown no elevation of temperature indicative of Coast fever can or should be removed to other clean areas. This method can even be simplified on an infected farm. The temperature need not necessarily be taken, as within twenty days most infected animals will show distinct symptoms of the disease. The interval between each new move should be about a month. The sick cattle should gradually be weeded out, and no more will sicken, if they be kept off infected areas. After a year of interval from the last death thereon the cattle could be put on to the land where infection started, as it will in the meantime have become clean. These are the practical deductions which one can make from the foregoing experiments.[1]

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[1] We have in our office records of outbreaks abundant evidence proving that infected pastures are not safe six months after the last animal has died.

II.—Do Salted Cattle Contain the Piroplasma Parvum in their Blood?

At the conference on cattle diseases held at Bloemfontein on 3rd, 4th, and 5th December 1903, Professor Koch stated that animals recovered from East Coast fever still retained the micro-organisms in their blood, which could only be detected by an expert by means of microscopic examination, and that these recovered animals continued to infect ticks on the pastures. The delegates from the Transvaal doubted the correctness of this statement because a very pertinent observation in the field of the Transvaal was then available, which showed that immune (recovered) and susceptible stock had grazed together with impunity for over nine months on clean ground. One of us (Theiler) has since shown that the forms of parasites which Professor Koch saw in oxen immune against Coast fever had nothing to do with that disease, since they were found in green susceptible cattle following injection of blood from oxen immune against ordinary Redwater, and which had never been in an area infected with East Coast fever. In addition to this observation may be quoted our experiment at Nelspruit, where, notwithstanding the fact that salted oxen were constantly grazing there, the farm became purified. It was evident that the question could easily be decided by experiments with brown ticks. It is now well known how easily they disseminate Coast fever, and that it is the adult brown tick which acts as the principal propagator.[2]

The tabulated form attached gives records of the origin, and the feeding of brown ticks on immune ox (salted) No. 101, which had been brought up from Nelspruit.

On representations from the delegates of the Transvaal and Portuguese Governments, who represented the only other territories then infected in addition to Rhodesia, the matter was considered by the conference as still *sub judice.* The conference, however, by a majority refused to accept Professor Koch's opinion that the disease was bound to go all over the country, and that therefore the wisest and most expeditious policy was to let it go, and trust to a method of immunisation which had then received but a short trial.

From the tabulated record it can be seen that ten different lots of brown ticks of various origin were utilised. The first stage of feeding as larvæ was passed on cattle susceptible to East Coast fever; the second as nymphæ was passed on the immune ox No. 101. The various adult brown ticks were collected and mixed.

Experiment 1.—On 25th August 1904, some of the above ticks were placed on ox B. 249. Further infestations took place on 30th August, 3rd, 7th, 13th, 15th, and 17th September. There were sixty-seven engorged females collected, and numerous males remained attached for several weeks. Ox B. 249 did not take East Coast Fever and is still alive.

Experiment 2.—Ox B. 149 was also infested in the same way on 7th Sept. 1904. The engorged females began to drop on 16th Sept. Eleven engorged females were collected. Several males remained

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[1] See also "Annual Report of the Principal Veterinary Surgeon for 1904." Eradication.

[2] See article by Theiler, "R.A.M.C. Journal," Dec. 1904.

No.	*Origin.*	*Date of Collection.*	*Egg laying Starts.*	*Hatching into Larvæ.*	*Feeding as Larvæ.*	*Dropping as Engorged Larvæ.*	*Moulting into Nymphæ.*	*Feeding as Nymphæ.*	*Dropping of Engorged Nymphæ.*	*Moulting into Adults.*	*Feeding as Adults.*
A	Nelspruit Sheep 8	21/12/03	10/1/04	9/2/04	23/2/04 on Texas Ox 224	29/11/04	26/3/04	5/5/04 on Komatie Ox 101	11/5/04	15/8/04	25/8/04 on Ox B 249
B	Nelspruit Ox 28	22/12/03	12/1/04	9/2/04	Do.	Do.	Do.	Do.	Do.	Do.	Do.
C	Station Ox 34	27/12/03	12/1/04	15/2/04	Do.	Do.	Do.	Do.	Do.	Do.	Do.
D	Nelspruit Ox 32	2/1/04	10/1/04	11/2/04	Do.	Do.	Do.	Do.	Do.	Do.	Do.
E	Station Ox 207	4/1/04	13/1/04	17/2/04	Do.	Do.	Do.	Do.	Do.	Do.	Do.
F	Station Ox 190	7/1/04	13/1/04	13/2/04	Do.	Do.	Do.	Do.	Do.	Do.	Do.
G	R. T. K. Oxen 29, 31	18/1/04	24/2/04	7/3/04	11/3/04 on Somali Ox 186	—	7/4/04	12/4/04 on Komatie Ox 101	16/4/04	24/6/04	25/8/04 on Ox B 249
H	Nelspruit Oxen 39, 43, 45	20/1/04	10/2/04	3/3/04	14/3/04 on Somali Ox 186	—	7/4/04	19/10/04 on Komatie Ox 101	22/4/04	24/6/04	25/8/04 on Ox B 249
I	Oxen 38, 39, 41, 42, 43, 46	23/1/04	8/2/04	7/3/04	18/3/04 on Somali Ox 186	—	7/4/04	19/4/04 on Komatie Ox 101	22/4/04	4/7/04	25/8/04 on Ox B 249
K	Nelspruit Ox 39	25/1/04	6/2/04	7/3/04	14/3/04 on Ox 709	19/3/04	3/4/04	5/5/04 on Komatie Ox 101	10/5/04	11/8/04	25/8/04 on Ox B 249

on the animal for some time afterwards. Ox B. 149 was still alive on 1st April 1905, and had never shown symptoms of the disease.

Experiment 3.—Some of the lot of ticks referred to under letter K. in the tabulated record were placed on ox B. 245 on 19th, 20th, and 21st September 1904. Only a few females were amongst these adults, which consisted principally of males, which were seen to be attached for a long time to B. 245. This animal was still alive on 1st April 1905, and had never been sick.

Experiment 4.—The brown nymphæ which were collected from the immune oxen exposed at Nelspruit on 13th and 24th August, 12th, 19th, and 26th September, 1st and 25th October 1904, moulted in the laboratory, and the adults were on 2nd November 1904 placed on Bull 257, where the males remained until the 21st November. Forty engorged females dropped and were collected. This bull was still alive on the 1st April 1905, and had never shown symptoms of disease.

CONCLUSION.

Taking into consideration that in the Pretoria[1] experiments with pathogenic brown ticks, the disease was produced in eight cases out of nine, and that only two ticks were necessary to cause the disease, the failure of the four experiments with ticks fed on salted oxen, two of the said experiments being carried out with a large number of ticks, has only one interpretation, namely, that the brown adults fed as nymphæ on cattle immune against Coast fever do not propagate the disease, and that therefore the blood of immune oxen does not harbour the parasite.

Reviews.

Annual Reports of Proceedings under the Diseases of Animals Acts, etc., for the year 1904. Board of Agriculture and Fisheries.

THE first part of this annual publication is, as usual, the report of the chief veterinary officer, Mr A. C. Cope, who at the end of the year under review retired from the department which he has served so long and so faithfully. It is a happy circumstance that Mr Cope, in what may thus be termed his valedictory report, is able to state that the country is now free from several of the more serious contagious diseases which were throughout the greater part of his period of service more or less rampant among the domesticated animals. During the year 1904 no case of pleuro-pneumonia, foot-and-mouth disease, or rabies occurred in Great Britain, and substantial progress was made in the eradication of swine fever, the total outbreaks of that disease for the twelve months having been 1196, as against 1478 in the immediately preceding year, and 5682 in 1894, when the disease was first taken directly in hand by the Board of Agriculture. There is then every encouragement to persevere with, and even to strengthen, the procedure which has proved so successful during the last few years, though the fact that in 1904 the disease occurred in no fewer than sixty-four countries indicates that it may be some years before its complete eradication is effected. It is interesting to observe that its persistance can hardly be ascribed to lack of co-operation on the part of the owners of pigs, for during the year no fewer than 7951 suspected cases were reported which the officers of the Board were unable to confirm.

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[1] Theiler, "R.A.M.C. Journal," Dec. 1904.

As an offset to the success of the Board in dealing with swine fever, it has unfortunately to be recorded that there was an increase in the reported outbreaks of anthrax, and in the number of horses attacked with glanders.

The assistant secretary, Mr Anstruther, contributes a report in which the proceedings of the Board in dealing with the contagious diseases are discussed from an administrative point of view. From this we learn that the continuance of the present ineffective measures against glanders is due to the refusal of the Treasury to contribute from the exchequer towards the expenditure which would be involved by the slaughter with compensation of all horses reacting to the mallein test. It is to be hoped that the pressure of public opinion, exercised through the local authorities, will ere long induce the Treasury to reconsider this decision. The report contains the usual array of interesting details set forth in statistical form.

Veterinary Diagrams, with Keys and Explanatory Notes, By Henry C. Wilkie, F.R.C.V.S., Veterinary Division, Department of Agriculture, New Zealand. Wellington: John Mackay, 1904.

THESE diagrams include the skeleton and foot of the horse, and the udder of the cow, mare, and ewe. Although apparently designed mainly for the instruction of agriculturists, horse owners, and shoeing smiths, they are calculated to be of service to veterinary students, as their large size gives them an advantage over the illustrations of the same subjects which are to be found in the text-books of anatomy. The diagrams may be obtained from Messrs Gay & Bird, Bedford Street, Strand, London.

Report.

THE ETIOLOGY OF HOG CHOLERA.[1]

By M. DORSET, M.D., B. M. BOLTON, M.D., and C. N. MCBRYDE, M.D., Biochemic Division, Bureau of Animal Industry, United States Department of Agriculture.

AFTER the fundamental work of Salmon and Smith relative to hog cholera and the corroboration of their observations by so many other investigators, it was regarded as settled beyond dispute that a certain micro-organism, B. choleræ suis, is the cause of hog cholera. Although certain results of Salmon and Smith, as well as the experience of others, show that it is difficult to reconcile all the facts as found with the acceptance of this bacterium as the exclusive factor in the production of the disease, more or less plausible explanations of these discrepancies have been offered; but these explanations did not seem entirely satisfactory, as will be shown later. Since this is the case, since the etiology is incompletely understood, it is difficult to attack properly the important practical questions of prevention and cure. In the present investigations we believe that, although we have by no means exhausted the subject, we have been able to make some very significant observations which throw light upon the perplexing question, and which we hope may eventually lead to a rational method of combating the disease.

In spite of the fact that the disease hog cholera is so well known, and that it has been so often defined and described in many excellent treatises, it will

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[[1] The complete report of this investigation has been published as Bulletin No. 72 of the "Bureau of Animal Industry," U.S.A. Department of Agriculture. In what is here reprinted the details of many important experiments are omitted. The researches are of the greatest possible interest to British veterinary surgeons, since it is generally admitted that American hog cholera and English swine fever are identical diseases.—Eds.]

nevertheless hardly be deemed superfluous to state at the outset just what our experience has taught us to regard as the essential features of the disease, on the one hand, and the variable and inconstant features, on the other. A definite statement in this regard is perhaps particularly called for, since all authorities do not agree upon the relative importance of the different manifestations seen in the disease. This discrepancy doubtless arises from the fact that in hog cholera, as in other diseases, in different outbreaks and at different times, one or the other set of symptoms and lesions predominate, and it is only by a study of various outbreaks in different parts of the country and over a long period of time that it is possible to arrive at an accurate knowledge of the variations to which the disease is subject.

Hog cholera is an acute and highly contagious disease which, so far as known, affects only hogs. It is characterised by fever, loss of appetite, weakness, and, where the affected animal does not quickly succumb, by rapid emaciation. Diarrhœa is usually present, though the affected animal may be constipated. Epistaxis frequently occurs, and there is often a gluing together of the eyelids. In the most virulent forms of the disease especially there may be ecchymoses in the skin over the ventral surface of the body. These are usually in the form of discrete splotches, but at times the entire ventral surface is diffusely reddened.

The changes seen in the internal organs vary greatly even in different animals in one and the same outbreak in a given herd. In general these lesions may be said to be either those of a hæmorrhagic septicæmia or of an ulcerative enteritis, the latter particularly pronounced in the cæcum and colon. The hæmorrhagic lesions are characteristic of the rapidly fatal form of the disease known as "acute hog cholera," the ulcerative intestinal lesions being especially prominent in those outbreaks where the animals do not succumb so rapidly; both the ulcerative and hæmorrhagic lesions may, however, be seen in the same animal. These two forms are not supposed to represent distinct diseases, but merely different types of the same disease, as already stated, the chronic form apparently resulting from either a decreased virulence of the infecting organism or a heightened resistance on the part of the animals which are attacked.

Hog cholera may be conveyed from sick to healthy animals almost if not quite without exception by contact, by feeding the viscera of diseased animals, and by the subcutaneous injection of the blood of sick animals. The disease may also be produced, according to Salmon and Smith, by feeding cultures of B. choleræ suis and by subcutaneous or intravenous inoculation of that organism. Welch and Clement claim that the disease may be produced by the intratracheal inoculation of healthy animals, as well as by intravenous inoculation with B. choleræ suis. Salmon and Smith found it very difficult to produce hog cholera by feeding or by subcutaneous injection of cultures of B. choleræ suis, but they consider that in certain instances they succeeded in doing so by both methods.

The mortality among hogs attacked by the disease varies from 30 to 100 per cent. of the affected animals, the death rate from the acute type of disease usually exceeding 80 per cent. of the affected herd.

The Infectiousness of the Blood of Animals Affected with Hog Cholera.—It will be noticed in the brief description that we have just given of hog cholera that the statement is made that this disease is communicated readily to healthy animals by inoculating them subcutaneously with the blood of hogs suffering with spontaneous hog cholera. The infectious nature of the blood has long been recognised, Law[1] having recorded experimental evidence of that fact in 1878. Other experiments showing the infectiousness of the blood of sick hogs are contained in the *Bureau of Animal Industry* report issued in 1889.[2] On p. 110 of that report Salmon and Smith describe the inoculation of four hogs subcutaneously with the blood of hogs sick of hog cholera, three of the four inoculated animals dying as a result. We would call attention here to the striking difference between the readiness in producing the disease by the inoculation of blood from animals sick from the spontaneous disease, on the one hand, and the great difficulty experienced in producing the disease by subcutaneous injections of pure cultures of B. choleræ suis, on the other. This and other important differences will be fully discussed later.

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[1] "Ann. Rep. U.S. Com'r. Agr.," 1878, p. 377.

[2] Hog cholera: Its history, nature, and treatment. Wash., D.C., 1889.

It is suggested by Salmon and Smith that these successful results may have been due to the greater virulence of the organism in the blood, but they also suggest the possibility that the injected blood coagulating in the tissues protected B. choleræ suis from the injurious effects of the body fluids and from the attacks of phagocytes, and at the same time furnished the injected organisms with an abundant supply of suitable food. The bacteria being thus protected for a time might gradually acquire sufficient powers of resistance to enable them to enter the circulation and produce the disease.

At the outset of our present investigations it was deemed wise to repeat Salmon and Smith's experiments with the blood of diseased animals, and to ascertain to what the pathogenic power of the blood was due. Accordingly, healthy hogs were inoculated subcutaneously with the blood of sick animals, the infection being derived from a number of separate and distinct outbreaks of hog cholera. We succeeded with the greatest regularity in communicating the disease to healthy animals by the subcutaneous injection of diseased blood obtained during the earlier stages of an outbreak. For convenience the blood was defibrinated previous to injection. It was also found that the clear serum which separated from the red cells in the bottles after the diseased blood was drawn under aseptic conditions possessed the power of inducing the disease in the same manner as the defibrinated blood. In the rare exceptions where the disease was not communicated in this way, it was found that the animal that was injected possessed immunity, as proven by subsequent exposure to infection in a pen with animals sick of hog cholera; there was no exception to this. The subcutaneous injection of blood from diseased animals proved indeed to be such a convenient and reliable method of transferring hog cholera from one animal to another that we have used it almost exclusively for propagating the several strains of disease with which our experiments have been conducted. But while it is true we have experienced no difficulty in thus propagating the disease with inoculations of blood, it should be explained that the different outbreaks which furnished the virus with which we have worked were selected because they appeared to possess a high degree of virulence. It is quite possible that in mild forms of the disease the same difficulty might be experienced in transferring the disease by blood inoculations as is found in transferring it by contact or by any other method.

What has been said above in regard to the result of inoculation with blood applies to blood obtained from animals at the beginning of an outbreak or during its height. Our own experience in propagating the disease by the successive inoculations of a number of hogs during a period of time covering several months has been that after a while the blood inoculations fail to cause the death of the animals. The virus seems to lose in potency after a number of passages through hogs.

The amount of diseased blood required to produce hog cholera when injected subcutaneously has not been definitely determined, but probably varies considerably, depending upon the virulence of the disease and the natural resisting power of the injected hog. We have produced the disease quite uniformly with from ½ to 2 cc. of blood, and in some instances with much smaller doses. For keeping the disease in existence, larger doses (5 to 10 cc.) have usually been employed. This was done in order to avoid the loss of the disease through a possible lowering in virulence, the dose in most instances being no doubt much in excess of the amount required.

Although we can not speak definitely, owing to lack of experimental data, we would not expect the chronic form of hog cholera to be so readily communicated in this way. The lesions in the chronic form indicate a tendency toward a localisation of the infectious centres in the intestines, whereas the acute form possesses the characters of a septicæmia.

In addition to proving the ease with which hog cholera may be transferred from one animal to another by the subcutaneous inoculation of blood, the inoculation experiments indicate that this infectious property is not the result of the mechanical protection of B. choleræ suis by the clotted blood, the possibility of which was suggested by Salmon and Smith, as previously stated. All of our blood inoculations were made after the fibrin had been removed, the defibrinated blood containing corpuscles and serum, or the serum alone being used. The serum was diluted with ten volumes of sterile bouillon before injection, and could not have afforded any mechanical protection from the phagocytes or other injurious agents. We must therefore look for some other explanation of the fact first observed by Salmon and Smith, namely, that hog cholera may be easily induced by subcutaneous injections of diseased blood, whereas the injections of pure cultures of B. choleræ suis in the same manner fail, in the great majority of cases, to produce disease.

The Contagiousness of the Natural Disease Hog Cholera.—In connection with the infectiousness of the blood of hogs affected with hog cholera, the readiness with which transmission of the disease takes place by simple contact with diseased animals, or by exposure in infected pens, must be taken into consideration. This high degree of contagiousness of hog cholera is universally recognised by the farmer as well as by those who have made experiments to test the matter; for, aside from the abundant evidence of contagiousness to be derived from the records of natural outbreaks of hog cholera, such as the accidental introduction of a sick pig into a healthy herd, we would call especial attention to certain records contained in the *Bureau of Animal Industry* report.[1] On pp. 145 to 153 inclusive, of that report will be found the records of a number of hogs that had been subjected to various methods of treatment, the object of the treatment being to render them immune from hog cholera. An examination of those records will show that the treated animals and controls were subsequently placed in infected pens to determine the value of the immunising agent, and that after such exposure to the natural disease, thirty out of a total of thirty-three that were exposed died of hog cholera. These figures include the check animals which were exposed with the treated ones.

We are in possession of the records of a large number of recent experiments which confirm in every way the evidence cited above, and we would emphasise the importance of this contagiousness as a necessary feature of the disease.

Immunity in Hogs which have Recovered from Hog Cholera.—Hogs that have recovered from natural attacks of hog cholera are immune from subsequent attacks. This fact is quite generally known among hog raisers, and causes the average farmer to place a higher value upon these so-called "cholera proof" hogs that upon non-immunes. During the course of experimental work in the past few years we have had the opportunity of observing the course of a large number of separate outbreaks of hog cholera, and have tested the immunity of a number of animals that recovered from these natural attacks by means of field exposure and by injections of diseased blood, but have never seen one of them succumb to a second attack. Immunity acquired in this way seems to be remarkably high in degree and very permanent. The same thing has been found to be true of hogs which have recovered from the disease produced by subcutaneous injections of hog cholera blood, such animals being found immune from hog cholera when exposed to the disease in a natural way and also when injected subcutaneously with blood from diseased animals.

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[1] Loc. cit.

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A point of especial interest which is fairly well brought out is that a severe attack of hog cholera is not necessary for the production of immunity from subsequent attacks. In fact, the protection derived from a mild attack appears to be quite as complete and lasting as that afforded by a severer form of the disease. That this immunity is not transitory is shown by the fact that we have been unable to induce the disease in some of these immunes by injections of diseased blood made nearly a year after recovery from the original attacks.

Recapitulation of the Characteristics of Hog Cholera.—Before proceeding to a description of the various methods we have adopted in our efforts to reproduce hog cholera artificially, we wish to bring together in concise form those characteristics which appear to be essential before any disease may be classified as hog cholera, without, however, offering them as a means of differential diagnosis.

(1) The lesions found at autopsy should be those commonly met with in either acute or chronic hog cholera, and which have been already fully described.

(2) The disease should be contagious, as proven by intentional exposure of healthy animals placed in the pen with sick ones or by the history of an outbreak.

(3) The blood of diseased animals should be capable of producing an attack of hog cholera when injected subcutaneously into healthy hogs.

(4) Animals which have recovered from an attack of hog cholera should possess immunity when exposed to natural infection or when injected subcutaneously with the blood of hogs suffering with hog cholera.

The lesions found at autopsy are variable. This variability of the pathological lesions is seen during the course of practically all outbreaks of hog cholera. In some animals there is almost complete absence of lesions; in others there may be few or many of the lesions so generally regarded as pathognomonic of that disease. The hog may be desperately sick and yet when killed the autopsy may reveal only a few reddened lymphatic glands and possibly a few hepatised areas in the lungs; or, on the contrary, every organ may show pathological changes. Without evidence of contagiousness and without the *post-mortem* examination of other hogs from the same herd, some of the animals could not be said to have died of hog cholera. On the contrary, the contagiousness, the infectiousness of the blood, and the acquired immunity, after recovery, are very constant features.

Experiments with Pure Cultures. — Having become impressed with the constancy of the three characters which were found in all natural outbreaks of acute hog cholera studied by us, and finding after an examination of the literature upon the subject that the questions of the contagiousness of the disease and the infectiousness of the blood in animals injected with pure cultures of B. choleræ suis had apparently not been made the subject of experiments in earlier investigations, we determined to make a certain number of experiments which would be in a way supplementary to those already recorded by Salmon and Smith and by Welch and Clement, and which would at the same time enable us to compare the results obtained with the blood and tissues of hogs suffering from hog cholera, on the one hand, with those obtained with cultures, on the other; in short, to see whether hog cholera with the three essential characteristics of contagiousness of sick animals, infectiousness of the blood of sick animals, and immunity when exposed to spontaneous infection after recovery, is found in the disease produced by cultures.

Experiments were undertaken with a view of obtaining light on these points. In making these experiments some hogs were injected subcutaneously, others intravenously, and others were fed.

The results of the subcutaneous inoculation of hogs with cultures of B. choleræ suis show that such inoculations produce serious disease in a relatively small number of cases.

Of the nineteen hogs injected subcutaneously, only three died. Salmon and Smith also encountered this difficulty in producing disease by subcutaneous inoculation, but seemed to regard this as being due to lack of virulence of the organism. This explanation is hardly satisfactory in our cases, for the organisms were very virulent for rabbits and guinea-pigs, and produced death in hogs with great uniformity when injected intravenously even in minute amounts, besides producing disease and death when fed to hogs in very small doses. So there can be no doubt as to the virulence of the cultures employed. The fact already brought out, that the injection of blood serum of hog-cholera hogs produces disease promptly and uniformly, suggested the trial of additions of normal defibrinated blood to cultures for subcutaneous inoculation; but the injection of cultures mixed with defibrinated blood proved to be as little efficacious in producing disease as the injection of cultures alone.

The intravenous injection of even very small amounts of culture produced fatal disease with great regularity, as already stated, and the feeding of cultures frequently caused sickness and death.

In these experiments, then, B. choleræ suis was constantly fatal to hogs when administered intravenously, and usually so when administered by mouth, but not as a rule when injected subcutaneously.

The symptoms produced by the organism when fed are those of intestinal disturbance mainly; the symptoms after intravenous injection are profound depression, loss of appetite, with death in a few days. The autopsy in both cases frequently shows hæmorrhagic lesions in the kidneys and other organs, and greater or less necrosis and even ulceration of the mucosa of the intestines, particularly of the cæcum in the region of the ileocæcal valve, the intestinal lesions being more marked where cultures are fed. So far, then, as symptoms and lesions are concerned, the disease in hogs produced by B. choleræ suis may very closely resemble those found in natural outbreaks of hog cholera. At least we are unable to make out any essential differences between the two diseases in these respects. But in certain other very important respects there exists a most striking difference; for, while the disease contracted by natural infection is highly contagious and the blood of animals sick from natural infection is almost always infectious for other hogs by subcutaneous inoculation, and, moreover, while hogs recovered from disease contracted in a spontaneous outbreak possess a high degree of immunity from subsequent infection, all these features are wanting in the disease produced by cultures of B. choleræ suis.

The lack of contagiousness in the disease produced by B. choleræ suis is rather surprising, for it would be natural to suppose that hogs made sick with the cultures would readily communicate the disease, particularly in those cases accompanied by the profuse diarrhœa which is often seen, notably in those where the hogs have been made sick by feeding cultures; yet in only one case—and that not free from objection—is there any indication that healthy hogs contract disease by associating intimately with hogs sick from cultures of B. choleræ suis. In this case the symptoms, which developed in seventeen days, were those of a passing sickness, shown by diminution of appetite and a gluing together of the eyelids, but all symptoms subsided in three or four days. This animal afterwards survived exposure in a pen with hogs sick of the natural disease. So, while it can not be asserted positively that this animal was not made sick by contact with the culture hog 207, there are several considerations which make this at least improbable, for a hog, 217, inoculated with blood drawn from the tail of the culture hog 207 remained well after the inoculation and did not acquire immunity, as was shown by the death of the hog in the exposure pen. Again, a hog, 221, fed with viscera of hog 207 became somewhat sick from eating the viscera, but also failed to acquire immunity. So, if of the two hogs, 217 and 221, the one inoculated with blood and the other fed with the viscera of hog 207, neither became immune, then it is not probable that hog 214 should have acquired immunity from simple association.

There were twenty-one hogs in all exposed to hogs which were given cultures of B. choleræ suis by different methods of administration, and hog 214 was the only one of the twenty-one to show any signs of sickness. Of these twenty-one hogs exposed to culture hogs, eighteen were subsequently placed in a pen with hogs suffering from natural hog cholera, and sixteen of the eighteen contracted the disease, showing that all except two of the eighteen hogs were susceptible and would have contracted the disease from the culture animals if these had been suffering from infectious hog cholera, as we understand the disease. Of the two hogs remaining well in the exposure pen, one, hog 214, has been discussed above, where it is shown that the animal probably did not derive immunity from the sickness contracted while in the pen with the hog made sick by inoculation with a culture, but in all likelihood possessed natural immunity. The most reasonable explanation of the failure of the other one of these hogs, 241, to become sick in the exposure pen is also that the animal had natural immunity, since it showed no evidences of sickness in the pen with the hogs inoculated with cultures, and one of these, 239, became sick and died in the exposure pen after recovering from the sickness produced by the culture. Hog 240, the companion of hog 239, died from the effects of the inoculation with the culture.

The injection of blood from a hog suffering from the natural infection is uniformly followed by the disease. This is the experience of all who have tested it, and we have abundant experimental proof of this fact. Indeed, we use this means of propagating the disease for experimental purposes in preference to any other on account of its certainty, as already stated. The injection of blood from animals sick from the administration of cultures, on the contrary, did not produce disease in any of the eleven hogs inoculated in this way. Of these eleven hogs, ten were exposed to natural infection, nine became sick, and seven died. This, again, is in marked contrast with the natural disease, for, in the rare instances of recovery after inoculation with the blood from a hog suffering from hog cholera, there is almost, if not always, without exception, the production of immunity.

Finally, the administration of cultures of B. choleræ suis seems to have little, if any, effect upon the production of immunity from natural infection. Incidentally, this has been shown above, in connection with the discussion of the power to produce disease with cultures; but it is worthy of more than passing mention, since it constitutes such a marked contrast with the results following recovery from the spontaneous disease, and from inoculations with blood from hogs suffering from the spontaneous disease.

There were eighteen hogs which survived the administration of cultures; sixteen of these were injected subcutaneously and two fed. Of these eighteen hogs, sixteen died, one became sick but recovered, one remained well when exposed to natural infection. Of these two which survived, both had previously been made sick by subcutaneous injection of cultures. Of the sixteen that contracted hog cholera and died in the exposure pen, six had recovered after being sick from subcutaneous injection of cultures, two from feeding, and eight had shown no sickness from the administration of cultures.

The difference between the disease produced by B. choleræ suis and spontaneous hog cholera, in respect to the immunity of hogs which recover, is very marked; for it can not be admitted that even the hog which failed to become sick in the exposure pen derived its immunity from the administration of cultures, since it may be claimed with equal probability that this animal had natural immunity. Indeed, it seems more probable that the latter explanation is correct in view of the many instances in which illness after administration of cultures failed to produce immunity.

It is clear, therefore, that while the disease produced experimentally with cultures of B. choleræ suis corresponds in some respects with spontaneous hog cholera, the two diseases show marked points of difference. They differ in contagiousness, infectiousness, and in the production of immunity from natural infection in animals that recover.

As an explanation of these differences between the natural disease and that produced by B. choleræ suis, the possibility that they could be accounted for by a sudden and profound alteration in the disease-producing properties of B. choleræ suis occurred to us. But this did not seem probable, for the reason that many cultures employed in the foregoing experiments were tested only a few days after isolation from the body of the sick hog; moreover, the same cultures proved to have retained full virulence for guinea-pigs and rabbits after cultivation for several months.

But if these conflicting results between the natural disease and that produced by B. choleræ suis are not due to a rapid change in virulence of the organism for hogs they must be due to the presence in the blood of some agent which is absent from the pure cultures, and which, acting either alone or in conjunction with B. choleræ suis, is able to bring on a typical attack of hog cholera.

In order to determine the presence or absence of this hypothetical virus in the blood of hogs sick of hog cholera, it became necessary to see whether we could obtain infectious blood from which B. choleræ suis was absent.

Two methods of obtaining such blood were finally adopted. The first was based upon the supposition that if two micro-organisms were concerned in the production of hog cholera they would probably not always invade the body simultaneously. Hence, by drawing blood from hogs at different stages of the disease we might be able to find a time during the illness when B. choleræ suis would be absent and the hypothetical virus would be present in the blood.

Experiments with Diseased Blood free from B. Choleræ Suis.—In order to obtain blood from the same hog on a number of consecutive days, we have used a very ingenious method which was devised by Dr W. B. Niles. In hogs the superficial blood vessels are either so small or are so situated that it is impracticable to use them for obtaining blood by the methods ordinarily employed. Dr Niles found that by cutting off the end of the tail an abundant supply of blood could be obtained, and, in addition, the blood drawing was comparatively simple and did not cause serious injury to the hog.

The general plan adopted for carrying out these so-called "tail-blood" experiments was as follows: A hog known to be healthy was injected subcutaneously with blood from a sick animal. Beginning forty-eight hours later, blood was drawn from the tail of the injected hog; a certain amount of the blood obtained in this way was injected subcutaneously into a healthy hog, and an equal quantity placed in neutral beef broth and incubated, the culture being made in order to determine whether or not B. choleræ suis was present. The object of these experiments was to determine whether the time at which the blood becomes infectious coincides with the appearance of B. choleræ suis, or whether this is independent of the appearance of that organism.

There was one contingency which it was feared might interfere with this experiment. In injecting the original hog with diseased blood the chances were in favour of B. choleræ suis being present in the injected blood. Under such circumstances it was feared that this organism would invade the body immediately and thus defeat our object. However, as no better method appeared available at the time, the experiments were conducted according to the plan described above, and it will be seen from the results that our fears were groundless, for B. choleræ suis did not appear in the general circulation until several days after the hogs were inoculated.

In the first experiment the blood was infectious on the second day, whereas B. Choleræ suis did not appear till the third day. In the second experiment the blood was infectious on the second day and B. choleræ suis did not appear until the fifth day. Beyond question, then, the blood of hogs suffering from hog cholera may be infectious for other hogs in the absence of B. choleræ suis, as is shown from the results given above. Even if we had only the results in these two cases this conclusion would not be unjustifiable, but when it is supported by the evidence of the filtration experiments below it becomes unavoidable.

We have laid the emphasis which the matter deserves upon the contagiousness of the natural disease, as contrasted with the lack of contagiousness in the disease produced by the use of cultures of B. choleræ suis, and it would complete these experiments with the tail blood taken from sick hogs intra vitam if it could be shown that hogs made sick by the injection of blood devoid of B. Choleræ suis could convey the disease to other hogs by association; but this matter was overlooked at the time the experiments were conducted. Still, since it will be shown in connection with the filtration experiments that those hogs inoculated with blood free from B. choleræ suis suffered from a highly contagious disease, it is but reasonable to suppose that the disease produced by the injection of tail blood, in all other respects resembling that produced by the filtered blood, should correspond with this in contagiousness as well.

Experiments with filtered Blood.—In addition to the experiments with the blood drawn from the tail during life, a second plan adopted was the filtration of diseased blood through Berkefeld and Chamberland cylinders. The filtration experiments were in part conducted before the tail-blood experiments; but for a better understanding of the results it has been deemed best to group all experiments of that character together.

It may be well, before entering upon a description of the filtration experiments, to explain briefly the conditions under which they were performed. First of all we would emphasise the fact that the most rigid precautions against accidental infection have been adopted in all cases. All pens had wooden floors. The dissinfection of pens and feeding troughs was in every instance most thoroughly carried out; the attendants in feeding the experimental animals never entered the pen nor was any implement used for cleaning the pens without thorough disinfection, both prior to and after use. The large majority of all hogs in these experiments were held in quarantine several weeks, and, where such a quarantine was impracticable, the animals were obtained from farms which where personally known to be free from disease. As a result of a rigid enforcement of these precautions there have been in the course of our experiments only a very few instances of disease that could be attributed to accidental infection. Any doubtful cases of this character have been left out of our report or else the conditions have been thoroughly explained.

In the foregoing pages it will be noted that we have insisted upon contagiousness as a necessary feature of hog cholera, and yet it will be found that in some of our filtration experiments pen checks were not used, and therefore the contagiousness of the disease in such instances was not established. In explanation of this fact it should be remembered that our conception of the disease hog cholera has been more or less modified by the results of our experiments; hence in the earlier work some details were omitted which were later found to be desirable. In work of this character, even with all possible facilities at our command, we have not failed to encounter difficulty in making each experiment absolutely complete within itself; but when the work is considered as a whole, we believe the sources of error will be found to have been eliminated so far as possible, and the results show very satisfactory uniformity.

The experiments have been made with eight distinct strains of disease. A part of the experiments were conducted at Washington, D.C., and a part at a temporary station in south-western Iowa. In obtaining virus from natural outbreaks for experimental purposes we have selected those instances of disease which showed unusual virulence and which would be classed as "acute hog cholera." Preceding each series of experiments will be found a short description of the outbreak from which the virus was obtained originally. The filters used were Berkefeld laboratory cylinders Nos. 5 and 7 (normal), and "Filtres Chamberland, Système Pasteur," marked "F" and "B," and all filtrations were, of course, conducted with sterile apparatus.

The serum was drawn off with a sterile pipette after the blood had stood for twenty-four hours or more in the refrigerator. The actual amount of serum used in most cases was 50 cc., though in some cases a smaller amount of serum was used, as it was not always possible to obtain 50 cc. free from red blood cells. The serum was then diluted with neutral beef broth or physiological salt solution in the proportion of 1 part of serum to 10 parts of the diluent, as stated in each case. The dilution was made for the reason that experience had taught us that the undiluted blood serum filtered very slowly, and in order to facilitate the passage of any virus that might be present. In most cases a fresh bouillon culture of about 4 or 5 cc. of B. prodigiosus was added to test the efficiency of the filtration. The vacuum employed varied from twenty-two to twenty-five inches, and the time of filtration was usually very short, about a half hour or less, though at times it was much longer on account of the presence of red blood cells. The apparatus was arranged in some cases so that the filtration took place from within the cylinder outward; in other cases from without inward.

The hogs used in the experiments were young animals weighing from 20 to 40 pounds, and the injections were made inside the thighs, using 22 cc.—11 cc. on each side—equivalent to 2 cc. of undiluted serum, except where otherwise stated.

Experiments with Blood from Herd I.—About the middle of February, 1903, the animals in this herd, which was located in south-western Iowa, began showing symptoms of illness. The farm was visited by Dr Charles M. Day, who reported that the *ante-mortem* symptoms were those of hog cholera. *Post-mortem* examinations of two of the sickest hogs revealed the lesions of acute hæmorrhagic hog cholera. As a result of this outbreak twenty-two out of twenty-three hogs died.

Blood from a sick animal in this herd was injected subcutaneously into hog 650, the latter animal being at once shipped to Washington. This hog, 650, served as the starting point for a large number of experiments. By successive subcutaneous inoculations of blood the disease was kept in existence for many months, no difficulty being experienced in transferring the disease to healthy animals in this manner, and the autopsies upon hogs which died from these injections were quite typical of hog cholera.

Filtration Experiment No. I.—A healthy hog, 704, was inoculated subcutaneously inside the left thigh with 4 cc. of defibrinated blood from hog 689, and this hog was sick at the time the blood was drawn, as a result of an injection of 5 cc. of diseased blood from a hog belonging to herd I.

Hog 704 remained well until six days after inoculation, when the first symptoms of illness were observed, and three days later, when the hog was very sick and weak, it was etherised and blood drawn from the heart. The autopsy showed a few punctate hæmorrhages in the kidneys and a few areas of congestion in the colon. The intestinal contents were bile-stained. The blood was defibrinated and kept on ice in a sterilized flask for one day, when the clear serum which had separated from the corpuscles was drawn off with a sterile pipette and diluted with physiological salt solution. A portion of the mixture was now filtered through a Chamberland cylinder "F."

The portion of the diluted serum which was not filtered was preserved for two days on ice, when it was filtered through a No. 7 Berkefeld laboratory cylinder, the conditions of filtration being the same as on the previous day when the Chamberland cylinder was used. In both of these filtrations, as well as in all others, every precaution was, of course, taken to prevent outside contamination, all flasks and filters being thoroughly sterilized before they were used. The culture in bouillon from the unfiltered serum of hog 704 was densely clouded twenty-four hours after being placed in the incubator. Upon plating from this flask a pure culture of a large organism resembling B. subtilis was found. B choleræ suis could not be detected.

The cultures made from the Chamberland and Berkefeld filtrates remained in the thermostat for three weeks without showing any signs whatever of bacterial growth.

Rabbit 739, which was inoculated with 10 cc. of the Berkefeld filtrate, died thirty-three days after inoculation. The autopsy disclosed no lesions whatever with the exception of echinococcus cysts in the liver. The echinococcus infection was no doubt the cause of death.

Rabbit 740, inoculated with the unfiltered serum, and rabbit 737 and guinea pig 3620, which were both inoculated with the Chamberland "F" filtrate, were all alive and well six months after inoculation.

Hog 749, inoculated with diluted, unfiltered serum, died thirty days after inoculation. There was no local inflammation at the point of inoculation. The inguinal glands were red and somewhat enlarged; spleen engorged with blood, slightly enlarged: kidneys congested, a few punctate ecchymoses in the cortical portion. In the small intestine and in the cæcum and colon a number of small erosions were found. The surface of these erosions was covered with a yellowish necrotic material which was easily removed. B. choleræ suis was obtained from the liver and spleen.

Hog 748, inoculated with Berkefeld filtrate, died nineteen days after inoculation. There was no inflammation at the point of inoculation. Inguinal glands enlarged and dark red; lungs showed two small areas of red hepatisation and a few small ecchymoses on the surface. This animal was otherwise normal with the exception of the kidneys, which had undergone a cystic degeneration, the parenchyma of these organs being almost wholly destroyed. Cultures taken from the liver and heart blood failed to reveal the presence of B. choleræ suis.

Hog 747, inoculated with Chamberland filtrate, died twenty-seven days after inoculation. There was no inflammation at the point of injection, but the areolar tissue in that region was stained a faint yellow colour. The right inguinal gland was considerably enlarged and quite red. The gland on the left side was much smaller and not so red. The liver exhibited on its surface a number of minute points of coagulation necrosis; spleen somewhat enlarged and dark; both kidneys presented a number of punctate hæmorrhages on their surfaces; in the neighbourhood of the ileocæcal valve in the cæcum and colon there was a number of comparatively recent ulcers. No cultures were taken from this animal because of *post-mortem* changes which where far advanced when the autopsy was made.

TABLE 15.

No. of Hog.	*Material Injected.*	*Inoculation*		*Autopsy.*	*Remarks.*
		Date.	*Result.*		
749	10 cc. diluted unfiltered serum hog 704	1903 May 27	1903 Died June 26	Hæmorrhages	B. choleræ suis present
748	11 cc. diluted filtered serum hog 704	Do.	Died June 15	Do.	Micrococcus from liver. B. choleræ suis not present.
747	Do.	Do.	Died June 23	Hæmorrhages and ulcers	No cultures taken.

Each of these hogs was kept in a separate pen.

In this experiment the features of especial interest are: (1) The absence of B. choleræ suis from the serum of hog 704, both before and after filtration, as determined by cultures and by the inoculation of small animals; (2) the production of fairly well-defined lesions of hog cholera in at least two of the hogs inoculated with this serum; (3) the failure of rabbits and guinea-pigs to succumb to doses of the serum which proved fatal to hogs; (4) the presence of B. choleræ suis in cultures from hog 749, although a culture test and the inoculation of a rabbit both indicated the absence of this organism from the serum with which the hog was inoculated.

Filtration Experiment No. 2.—Hog 751 was inoculated with 5 cc. of defibrinated blood from hog 703. Hog 703 was carrying the herd I. disease and was quite sick at the time blood was drawn. Hog 751 showed decided symptoms of illness fifteen days after inoculation and became gradually worse. Eighteen days after inoculation the animal was etherised and blood was drawn and defibrinated. The lesions found at autopsy were not marked, and did not seem sufficiently grave to account for the severe illness of the animal at the time it was killed. The spleen was enlarged and soft. The mucosa of the cæcum and colon was congested and exhibited a number of small erosions, and the intestinal contents were bile stained. The other organs were normal in appearance.

The defibrinated blood, which was collected in a sterile bottle, was kept on ice for two days, when the clear serum that had separated from the red cells was withdrawn with a sterile pipette, and diluted with sterile physiological salt solution. After reserving a portion of the diluted unfiltered serum the remainder was divided into three parts and filtered as follows: One portion was filtered through a sterile Berkefeld laboratory cylinder No. 7 (the filtrate was designated "751 Bd"); a second portion of the diluted serum was passed through a sterile Chamberland cylinder marked "F" (filtrate designated "751 Ch. F"); the third portion was passed through a sterile Chamberland cylinder marked "B" (filtrate designated "751 Ch. B"). In the case of the Chamberland filtrations the cylinders were arranged in the manner shown in Plate 2. From each of these three filtrates 25 cc. was removed by means of a sterile pipette and added to a flask containing 400 cc. of neutral beef broth. At the same time, 2 cc. of the undiluted serum of hog 751 was added to 400 cc. of sterile beef broth. All four of these flasks were placed together in a thermostat at 37·5° C.

The flasks inoculated with the unfiltered serum and with the Chamberland "F" filtrates became clouded, while those inoculated with the Berkefeld and Chamberland "B" filtrates remained clear after being in the incubator for nine days. An examination of the culture from the unfiltered serum showed it to contain a micrococcus in pure culture, while the flask inoculated with the Chamberland "F" filtrate contained a pure culture of a large bacillus which resembled B. subtilis. No attempt was made to identify these organisms, the examination being carried only so far as was necessary to ascertain that B. choleræ suis was absent. The presence of the large bacillus in the culture from the Chamberland "F" filtrate, in view of its absence from the unfiltered serum, can only be accounted for by accidental contamination from the air, for which there was abundant opportunity during the removal of the 25 cc. used for the culture.

Hog 766 was inoculated with defibrinated blood of 751 immediately after it was drawn, and placed alone in a pen; was first noticed to be sick in eleven days, and died in fourteen days. The autopsy revealed numerous small hæmorrhages and some œdema in the lungs. The liver and spleen were enlarged and congested; the kidneys hæmorrhagic. There was considerable pigmentation, and there were also numerous small hæmorrhages in the intestine; a few recently formed ulcers in the colon.

Hog 788 was inoculated with diluted, not filtered, serum and placed alone in a pen; was first noticed to be quite sick in nine days, and remained so for eighteen days, when improvement began. This improvement continued for thirty days, when the animal had apparently completely recovered, so it was placed in the exposure pen to test its immunity. The hog was killed after remaining in the exposure pen for about seven months, where other hogs were dying of hog cholera. It was in an unthrifty condition, but no lesions of hog cholera could be found.

Hog 783 was inoculated with Berkefeld filtrate; was first noticed to be sick in eight days. This hog was in a moribund condition when killed fifteen days after inoculation. The legs and ears were reddened and there were many red areas over the ventral surface of the body. There was no lesion at the point of inoculation. The lymph glands were œdematous and there were several areas of red collapse in the lungs. The kidneys were hæmorrhagic; the intestines appeared normal, although their contents were bile-stained.

Hog 789, inoculated with Berkefeld filtrate, showed symptoms of illness in eight days and died in eleven days. There was extreme reddening of the ventral surface of the body and of the head and ears; the lungs were sprinkled throughout with ecchymoses varying from 3 to 15 mm. in diameter; spleen enlarged and engorged with blood; kidneys presented a few punctiform hæmorrhages; some ecchymoses and ulcers in the mucous membrane of the intestines. Hog 783 and 789 were placed in a pen by themselves.

Hog 790, inoculàted with Chamberland "F" filtrate, was slightly sick in eleven days and became gradually worse and died twenty-eight days after inoculation. The ventral surface of the body was extensively reddened; the lymphatic glands were enlarged and red; the kidneys hæmorrhagic, and cæcum and colon extensively ulcerated.

Hog 791, a mate to hog 790, was injected on the same date with same lot of serum; was seen to be sick in seven days, became rapidly worse, and died in twelve days. There was extreme reddening of the ventral surface of the body and of the ears; no lesion at the seat of infection, but the adjacent lymph glands were enlarged and hæmorrhagic; the lungs were sprinkled throughout with ecchymoses; the spleen enlarged and congested; the kidneys presented punctiform hæmorrhages in the cortices; both large and small intestines were flecked with hæmorrhages of varying size, but no ulcers could be found. Hogs 790 and 791 were placed in the same pen.

Hog 784, inoculated with Chamberland "B" filtrate, became sick six days after inoculation and died in twelve days. The autopsy revealed lesions which belong exclusively to the hæmorrhagic type of disease. There was extensive reddening of the skin, reddeding and swelling of the lymphatic glands, but no inflammation at the seat of injection. Ecchymoses of varying size and number were present in the lungs, spleen, liver, kidneys, stomach, and large and small intestines.

Hog 786, inoculated at the same time and in the same manner as hog 784, became slightly sick eight days after inoculation, and being hopelessly sick on the fifteenth day, was killed. The lesions found belonged to the chronic intestinal type, and therefore differed quite markedly from those met with in its mate, hog 784. The only explanation of the wide difference in the lesions found in these two hogs seems to lie in a variation in resisting power possessed by the two animals. Hog 784 and 786 had been placed in a pen by themselves.

TABLE 16.

No. of Hog.	*Material Injected.*	*Inoculation.*		*Autopsy.*	*Remarks.*
		Date.	*Result.*		
		1903	1903		
766	5 cc. defibrinated blood hog 751	June 15	Sick June 23; died June 29	Hæmorrhages	No cultures taken.
788	20 cc. unfiltered serum hog 751	June 18	Very sick; recovered	—	Immune when exposed subsequently
783	20 cc. serum 751 "Bd."	Do.	Sick June 26; killed July 3	Hæmorrhages	In dying condition when killed.
789	Do.	Do.	Sick June 26; died June 29	Do.	No cultures taken.
790	20 cc. serum 751 "Ch. F."	Do.	Sick June 29; died July 16	Hæmorrhages and ulcers	Do.
791	Do.	Do.	Sick June 25; died June 30	Hæmorrhages	Do.
784	20 cc. serum 751 "Ch. B."	Do.	Sick June 24; died June 30	Do.	Do.
786	Do.	Do.	Sick June 26; killed July 3	Ulcers	In dying condition when killed.

The results of Filtration Experiment No. 2 are even more striking than those obtained in Filtration Experiment No. 1. Flasks inoculated with 25 cc. of the diluted serum—5 cc. more than was employed for inoculating a hog—failed to reveal the presence of B. choleræ suis in either the filtered or unfiltered serum. It seems extremely improbable that B. choleræ suis could have been present in all three of the filtrates and also in the unfiltered serum in sufficient numbers to produce acute hog cholera by subcutaneous injection and yet have failed to grow in the cultures from any of them. The possibility of accidental infection from without or from the pens or syringes also seems to be highly improbable in view of the uniformity of the results, and the great rarity of such accidental infection in other experiments where hogs were inoculated with substances other than the blood or tissues of hogs sick of hog cholera.

Notwithstanding the unlikelihood of the disease in these hogs having been caused by any other agent than the injected serum, plans were made for another set of experiments which would eliminate still further the chances of error mentioned above.

Filtration Experiment No. 3.—Hog 797, the animal which furnished the blood for this experiment, was inoculated subcutaneously with 5 cc. of blood from hog 792, which was carrying herd I. disease. Hog 797 became very sick eleven days after inoculation, and was etherised and blood drawn from the heart and defibrinated. The autopsy on hog 797 disclosed hæmorrhagic kidneys and a number of recent ulcerations in the cæcum and colon.

The blood obtained from hog 797 was kept on ice for three days. The serum was then withdrawn with a sterile pipette and mixed with sterilised physiological salt solution. Different portions of this diluted serum were then filtered through three sterilised filters—a Berkefeld laboratory cylinder No. 7 (filtrate designated "797 Bd."), a Chamberland cylinder "F" (filtrate designated "797 Ch. F."), a Chamberland cylinder "B" (filtrate designated "797 Ch. B.").

The Chamberland cylinders were so arranged that the filtration should take place from within outward; the Berkefeld cylinder was arranged in the usual manner. After completing the several filtrations, cultures were made as follows:—

To flasks containing about 400 cc. of sterile neutral bouillon 25 cc. of each of the filtrates was added, and to a similar flask 4 cc. of the original undiluted serum. All cultures were placed immediately in the incubator at 37·5° C. Three days after these cultures were prepared, the one made from the undiluted and unfiltered serum showed a dense clouding, and the cultures from the Chamberland "F" and "B" filtrates were both slightly cloudy. The culture from the Berkefeld filtrate remained clear. In order to ascertain whether B. choleræ suis was present in either of these cultures, guinea pigs were inoculated with $\frac{1}{2}$ cc. of each. The guinea-pig inoculated with the culture from the unfiltered and undiluted serum remained well. The same is true of the guinea-pig inoculated from the Chamberland "B" culture. The one inoculated with the Chamberland "F" culture died twenty-five days after inoculation. A careful bacteriological examination, however, failed to reveal the presence of B. choleræ suis, but indicated that death was due to infection by an entirely different organism.

Immediately after defibrination the blood of hog 797 was injected subcutaneously into hogs 819 and 820, 5 cc. into each. The inoculations with the diluted serum of hog 797 were made after the serum had been in the refrigerator overnight.

The numbers of the hogs concerned and their arrangement in the pens are as follows:—

[TABLE.

Hog 819.	Received 5 cc. defibrinated blood of hog 797	Placed in same pen.
820.	Do.	
837.	Received 22 cc. diluted, unfiltered serum of hog 797	Do.
827.	Uninoculated pen check	
832.	Received 22 cc. serum hog 797, filtered through Berkefeld	Do.
834.	Do.	
830.	Uninoculated pen check	
828.	Received 22 cc. serum hog 797, filtered through Chamberland "F"	Do.
829.	Do.	
836.	Uninoculated pen check	
824.	Received 22 cc. serum hog 797, filtered through Chamberland "B"	Do.
833.	Do.	
838.	Uninoculated pen check	
825.	Received in same manner, 22 cc. sterile bouillon	Do.
826.	Do.	
831.	Uninoculated pen check	

Hog 819 was inoculated with 5 cc. of defibrinated blood of hog 797; was desperately sick in eight days and was etherised in nine days in order that blood might be drawn for other experiments. The autopsy revealed minute hæmorrhages in the lungs, enlarged spleen, congested kidneys; intestinal contents were bile-stained, but no ulcers could be found.

Hog 820 was inoculated at the same time and with the same material as hog 819. This hog became desperately ill and was etherised nine days after inoculation in order that blood might be obtained for other experimental work. The autopsy was quite similar to that of hog 819, the only noticeable difference being that in the case of hog 820 the intestines were congested and some hæmorrhages were found on the mucous coat.

Hog 837 was inoculated with diluted, not filtered, serum of hog 797; showed no symptoms of illness for seven days, when it appeared to be very sick, and the symptoms increased in severity till death, eleven days after inoculation. There was no inflammation at the point of inoculation. The inguinal glands were slightly reddened; there were a few small hæmorrhagic areas in the lungs. The spleen was enlarged and dark; a few punctiform hæmorrhages were seen in the kidneys. The intestinal contents were fluid and faintly tinged with blood. The mucous membrane of the cæcum and colon presented a few small ecchymoses, and in the cæcum some small ulcers were found, evidently of recent formation. From the spleen of this animal a pure culture of a micro-organism resembling B. choleræ suis in all of its cultural characters except in its action upon saccharose was obtained. This sugar underwent fermentation with the production of gas.

Hog 827 was not inoculated, but placed as a check in the pen with hog 837; showed no signs of illness for fourteen days, when symptoms similar to those seen in 837 developed, and the hog died four days later. The subcutaneous areolar tissue over the abdomen and thorax was sprinkled with small ecchymoses. The inguinal, lumbar, mesenteric, and mediastinal glands were enlarged and reddened. Numerous petechiæ on the surface of the heart muscle. The spleen was somewhat enlarged and quite dark; kidneys were dotted with numerous punctiform hæmorrhages; the peritoneum contained a number of discrete hæmorrhages. A few small hæmorrhages were found on the serous surface of the stomach, and also on the serous and mucous surfaces of the small intestines, cæcum, and colon. No ulcers could be found. Cultures from the spleen revealed the presence of B. choleræ suis.

Hog 832 was inoculated with the Berkefeld filtrate from serum of hog 797; was quite sick in seven days and died in twelve days. There was no lesion at the seat of injection. The lungs were sprinkled throughout with minute hæmorrhages. The spleen was greatly enlarged and engorged with blood; the kidneys thickly sprinkled with punctiform hæmorrhages. The small intestine exhibited numerous circumscribed hæmorrhages in the mucosa. The mucosa of the cæcum and colon contained many small ulcers and the intestinal contents were blood-stained. No cultures were taken.

Hog 834 was injected with the same material at the same time as hog 832; was quite sick seven days after inoculation, became rapidly worse, and died on the twelfth day. There were no lesions at the seat of injection. The inguinal glands were enlarged and reddened; there were a few small ecchymoses on the surfaces of the lungs; the spleen was enlarged and congested; the kidneys were also congested and in addition were dotted with minute sharply defined hæmorrhagic points. A few superficial erosions were found on the mucous surface of the cæcum and colon. Cultures from the spleen revealed the presence of B. choleræ suis.

Hog 830 was not inoculated, but served as a pen check on hogs 832 and 834; seemed to be slightly sick in eleven days; was desperately sick in fifteen days and was killed. There were no lesions at the seat of inoculation. The inguinal, mediastinal, and mesenteric glands were all enlarged and very red. There were diffuse hæmorrhages in the epicardium and a large number of minute, sharply defined hæmorrhages on the surfaces ef the lungs. The spleen was very much enlarged; the kidneys showed the characteristic punctiform hæmorrhages in their cortices. The peritoneum was dotted with numerous small ecchymoses, as were the serous surfaces of the stomach, cæcum, and colon.

Hog 828 was inoculated with Chamberland "F" filtrate from serum of hog 797; first sick in seven days and was found dead in twelve days. The inguinal glands were somewhat reddened. On the surfaces of the lungs a few small hæmorrhagic points could be seen. The spleen was enlarged, cæcum and colon congested, and on the mucous surface certain small areas were covered with a tenacious yellow exudate which, upon being removed, revealed a rather superficial erosion of the mucosa.

TABLE 17.

No. of Hog.	*Material Injected.*	*Inoculation.*		*Autopsy.*	*Remarks.*
		Date.	*Result.*		
		1903	1903		
819	5 cc. defibrinated blood hog 797	July 14	Sick July 22; killed July 28	Hæmorrhages	No cultures taken.
820	Do.	Do.	Do.	Do.	Do.
837	22 cc. unfiltered serum hog 797	July 20	Sick July 27; died July 31	Do.	Atypical B. choleræ suis from spleen.
827	Not inoculated; pen check on hog 837	Exp'd July 20	Sick Aug. 3; died Aug. 7	Do.	No cultures taken.
832	22 cc. serum hog 797 "Bd"	July 20	Sick July 27; died Aug. 1	Do.	Do.
834	Do.	Do.	Do.	Do.	B. choleræ suis from spleen.
830	Not inoculated; pen check on hogs 832 and 834	Exp'd July 20	Sick July 31; killed Aug. 3	Do.	Do.
828	22 cc. serum hog 797 "Ch. F."	July 20	Sick July 27; killed Aug. 1	Hæmorrhages and ulcers	No cultures taken.
829	Do.	Do.	Do.	Do.	B. choleræ suis from spleen.
836	Not inoculated; pen check on hogs 828 and 829	Exp'd July 20	Sick Aug. 1 died Aug. 6	Do.	No cultures taken.
824	22 cc. serum hog 797 "Ch. B."	July 20	Sick July 27; died Aug. 1	Do.	B. choleræ suis from spleen.
838	Do.	Do.	Sick July 27; died July 31	Do.	Atypical B. choleræ suis from spleen.
833	Not inoculated; pen check on hogs 824 and 838	Exp'd July 20	Sick Aug. 3; died Aug. 11	Do.	B. choleræ suis from spleen.
825	22 cc. sterile bouillon	July 20	Remained well	—	—
826	Do.	Do.	Do.	—	—
831	Not inoculated; pen check on hogs 825 and 826	Exp'd July 20	Do.	—	—

Hog 829 was treated in the same manner as hog 828; first signs of sickness in seven days; was found dead in twelve days. The autopsy was almost identical with that of hog 828. Cultures from the spleen revealed B. choleræ suis.

Hog 836 was not inoculated, but served as a pen check on hogs 828 and 829. The first symptoms of illness were noted in twelve days and the animal died in seventeen days. A few small hæmorrhages were found on the auricular appendages of the heart. The spleen was enlarged and quite dark; the kidneys were very much congested and exhibited some punctiform hæmorrhages on their surfaces. Some small petechiæ on the serous surface of the stomach. The small intestine presented on its serous surface a large number of small, rather sharply defined ecchymoses. The serous surfaces of the cæcum and colon were less affected than the same portion of the small intestine. The mucous coat of the latter presented many small erosions, the centres of which were of a reddish colour. The contents of the cæcum were semi-fluid and contained some blood.

Hog 824 was inoculated with Chamberland "B" filtrate from serum of hog 797; was first noticed to be sick in seven days; became rapidly worse and died twelve days after inoculation. The entire ventral surface of the body was reddened. The inguinal, lumbar, and mesenteric glands were enlarged and red. There were numerous scattered hæmorrhagic areas on the surfaces of the lungs and the cephalic lobe of the left lung was hepatised. The spleen was enlarged and dark with irregular diffuse hæmorrhagic areas on its surface. The kidneys were thickly dotted with punctiform hæmorrhages. There were numerous small petechiæ on the serous and mucous surfaces of both the large and small intestines. The mucous membrane of the cæcum and colon contained a number of small ulcers.

Hog 833 was inoculated at the same time with the same material as hog 824; was quite sick in seven days and became rapidly worse, dying twelve days after inoculation. There were no lesions found at the seat of inoculation. The inguinal glands were enlarged and red, and there were many sharply defined hæmorrhages on the surfaces of the lungs; the spleen was somewhat enlarged; the kidneys congested and dotted with numerous punctiform hæmorrhages. Some hæmorrhages were found on the surfaces of the stomach and also on the serous and mucous surfaces of the intestines. The intestinal contents were blood-stained.

Hog 838 was not inoculated, but placed as check in the pen with hogs 824 and 833. The animal remained well for fourteen days, when it became suddenly ill and died twenty-two days after exposure. The lungs contained, besides several areas of red collapse, numerous small hæmorrhages. Both of the auricles of the heart were sprinkled with petechiæ. The spleen was enlarged, kidneys hæmorrhagic, and innumerable minute hæmorrhages were found on the mucosa of the small intestine; a number of ulcers were present in the mucosa of the cæcum and colon. Cultures from the spleen contained pure growths of B. choleræ suis.

Hogs 825, 826, and 831: Two of these hogs, 825 and 826, were injected with sterile bouillon, the other, 831, serving as a check. All three remained perfectly well for forty days. Hogs 825 and 826 were then injected with blood from a hog sick with herd II. disease, hog 831 being again used as a check. All three died, thus showing that their failure to contract disease when used in Filtration Experiment No. 3 was not due to natural immunity.

It is evident that the results obtained in Filtration Experiment No. 3 substantiate completely the work described under Filtration Experiments Nos. 1 and 2. B. choleræ suis could not be found in any of the material used for injection either before or after filtration, and yet the hogs inoculated with this material sickened promptly and the majority of them died, typical lesions of hog cholera being found in every instance. Filtration Experiment No. 3 was of greater value than the two preceding experiments on account of the check animals which were used in connection with the different inoculations. A glance at the accompanying table will show that in every instance the injected animals became sick from four to seven days sooner than the accompanying pen checks, thus indicating in a direct way that the infection arose from the injected material and not from the pens in which the animals were kept. Hogs 825 and 826, which were handled in the same manner as the remainder of the animals in this experiment, but which were injected with sterile bouillon instead of with the filtered diseased serum, remained perfectly well, thus furnishing proof that the disease in the hogs which sickened could not be attributed to an infection through careless handling or through the introduction of diseased material into the pens subsequent to inoculation. We would call especial attention to the fact that the subcutaneous injections of filtered blood not only caused the death of the injected animals, with typical lesions of hog cholera, but that the disease thus produced was contagious for susceptible animals placed in the pen with those which were injected.

Discussion of Filtration Experiments.—The results of the subcutaneous injections of hog cholera blood filtered through Chamberland and Berkefeld cylinders show that these injections produced disease quite as uniformly as those with the unfiltered blood. On the whole, it is true, hogs inoculated with the unfiltered blood became sick more quickly after inoculation than the hogs inoculated with the same serum after filtration, but this was not always the case. It is also true that a greater precentage of deaths occurred among the hogs inoculated with the unfiltered serum than among the hogs inoculated with the filtered serum. Of the thirty-six hogs inoculated with the unfiltered serum, all became sick, twenty-four died, ten were killed in a moribund condition, and two recovered; while of the sixty-one hogs injected with filtered serum, fifty-eight became sick, thirty-nine died, ten were killed in a moribund condition, nine recovered, and three showed no preceptible sickness. So although the mortality was less in the hogs injected with filtered serum, there was practically no difference in the production of disease in the case of unfiltered and of filtered blood; for the three hogs which failed to show noticeable symptoms of sickness after the injection of filtered blood must have possessed great resisting power, since two of them remained well on exposure to natural infection, and the other lived for many weeks in contact with sick hogs but died eventually, though it is not certain that death was due to hog cholera.

The resisting power of these three hogs may have been natural, or, as will appear from what is said later on, it may have been acquired from the serum injections. The possibility that they may have been really sick, but not have visible evidence of this, should not be lost sight of.

It is not surprising, however, that the blood after filtration should show less potency than blood before filtration; for whatever the nature of the infectious agent, it is but natural to suppose that some of it is removed by the filter. Nevertheless, diminution of potency is not observed in all cases, as already remarked, and when no diminution is observed it is probable that there is such a large amount of the infectious agent present originally, that although a part is removed by filtration, there still remains a sufficient amount to cause disease.

It will be observed that these results show not only that the filtered blood produces disease with great regularity, but that hogs which recover from the disease produced in this way have immunity from natural infection. Of the nine hogs that recovered after being sick, only one died in the exposure pen, but from the history of this case it seems probable that this hog, 1137, may not have fully recovered from the sickness produced by the filtered serum.

The results furthermore show that the disease pruduced by injection of filtered blood is contagious; for uninoculated hogs placed in the pens with the hogs inoculated with the filtered blood remained well until the inoculated animals became sick, and then contracted disease more or less promptly.

work has prevented their completion. In a few preliminary examinations, guinea-pigs and rabbits were inoculated subcutaneously with small amounts of the intestinal contents of normal hogs and a few agar plates were made from the same material. In one case only, when the method of Drigalski and Conradi for the isolation of typhoid bacilli was used, did we succeed in discovering an organism which possessed the cultural characters of B. choleræ suis. The only way in which this organism from the normal intestinal contents differed from the classical type of B. choleræ suis was in its lack of pathogenic power for guinea-pigs. It will be readily understood that the isolation of B. choleræ suis from material containing an immense number of colon bacilli is a matter of unusual difficulty, and it is therefore not surprising that we should fail to discover it even though it may have been present in very considerable numbers. The isolation of a non-virulent form of B. choleræ suis from the intestines is, therefore, considered very important on account of the support it lends to the hypothesis that B. choleræ suis may be frequently an inhabitant of the intestine of the normal hog.

If such a hypothesis be true, the conditions would be entirely analogous to those under which many pathogenic micro-organisms exist, as, for example, the pneumococcus in the mouths of healthy individuals, and the swine plague bacillus (B. suisepticus) on the tonsils of healthy hogs, and in the pens where healthy hogs are kept, the supposition being that micro-organisms present in small numbers or possessing a low degree of virulence are successfully resisted until certain influences either heighten the virulence of the bacteria or lower the resisting power of the animal body. The production of disease by feeding small quantities of B. choleræ suis might seem to indicate that that organism could not remain in the hog's intestine without giving rise to more or less serious disturbance, but it should be remembered that these small quantities of culture ($\frac{1}{10}$ to $\frac{1}{2}$ cc.) contain enormous numbers of bacilli and probably many more than the hog under ordinary circumstances is able to resist.

The toxin present in artificial cultures may also play some part in causing illness when cultures are fed, or it may be—and this last supposition seems not unlikely—that B. choleræ suis, if it exists in the bodies of normal hogs, possesses a very low degree of virulence, and that it is only through a lowered resistance on the part of the hog that it is enabled to enter the circulation and cause disease. The isolation from a healthy hog of an organism indistinguisable from B. choleræ suis except by absence of pathogenic power for guinea-pigs lends strength to the explanation last mentioned of the way in which hogs are liable to attacks by B. choleræ suis. B. coli communis is a familiar example of an organism constantly present in health, and yet assuming under certain conditions very great pathogenic power.

Whatever may be the port of entry or the influences which bring about an invasion of B. choleræ suis, we are convinced that the filterable virus was responsible for the high degree of infectiousness and, therefore, for the spread of the disease encountered in the several outbreaks of acute hog cholera described in previous pages of this bulletin. It seems reasonable to suppose that what has been found to be true in these eight outbreaks is also true of other epidemics of acute hog cholera, and therefore that the extensive losses occasioned by outbreaks of that disease are to be attributed, in the main, to the filterable virus. In many of the individual hogs, the fatal termination may have been in large measure due to B. choleræ suis, but the probabilities are that, without the filterable virus, comparatively few hogs would have been attacked.

It follows from all that has been demonstrated in the preceding pages that, if a practical method of protecting hogs from the filterable virus should be discovered, the problem of combating hog cholera, at least the highly infectious form of that disease, will have been solved.

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RINGWORM IN DOMESTIC ANIMALS.[1]

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THE forms of dermatomycosis or dermatophytis usually referred to under the general term "ringworm" are dermatoses produced by lowly-organised fungi known as epiphytes, ectophytes, or dermatophytes, and grouped by specialists into a small family — the trichophyta.

The trichophyta are usually included in the order of hyphomycetes or mucedineæ, in common parlance, "moulds." These humble fungi, consisting simply of filaments, fructify by means of a kind of bud thrust out by the filament and known as a conidium. Careful observation has shown, however, that certain mucedineæ are only lowly or conidial forms of more highly developed fungi, and it is even open to question whether the same is not true of all the hyphomycetæ.

Regarding the dermatophytes, Matruchot and Dassonville's studies of the orders they comprise and of those which exactly correspond to the family of gymnoasceæ, order ascomycetes, have resulted in their including in the latter the ectophytic fungi of animals. It is, however, well to bear in mind, as qualifying this conclusion, that the formation of asci (characteristic of ascomycetes) has only been detected in one species of dermatophytes, viz., *eidamella spinosa* of the dog.

The order of ascomycetes is distinguished from the other orders of the class of fungi (1) by its thallus or mycelium, which is septate, *i.e.*, divided by partitions, formed by successive cells, and (2) by the formation of spores in the interior of a cell named an ascus.

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[1] Translated from the "Revue Vétérinaire," Nos. 3, 4, and 5, 1905.

Among the ascomycetes, the family of gymnoasceæ is characterised (1) by the position of the asci, which are contained in a closed cavity, and (2) by the form and structure of this cavity, which is more or less spherical and is surrounded by entwined filaments which do not form a true membrane.

The dermatophytes multiply by the increase of their mycelium and by spore formation. The way in which the thallus grows causes them to form on the surface of the skin more or less regular circles and sometimes crusts depressed at the centre.

In its parasitic state a dermatophyte is formed of filaments of mycelium or tubes, limited by an amorphous membrane and containing a more or less granular protoplasm. Sometimes this protoplasm is continuous and the tubes form the growing part or thallus of the fungus; the cells are then much longer than wide. In other cases they are as wide as they are long and form what is known as mycelial spores. These spores may be rounded in form and by separating extend the area invaded by the parasite.

In cultures one finds spores formed by lateral budding; these are more characteristic of the species and are termed conidia. Such spores may also be found in the parasitic forms.

The difference between filaments and spores merely results from the stage at which they are studied. By elongating and developing the spore forms a filament of mycelium, in the interior of which the protoplasm develops, throws out lateral prolongations, and undergoes segmentation. The sheath in its turn becomes segmented and imprisons the segments of protoplasm; new spores are thus produced which develop in the same way. The whole filament does not invariably undergo this process, which is always most active at the extremities. Moreover, many filaments prove sterile and wither up, sometimes after having undergone partial segmentation.

The discovery of the parasitic nature of ringworm was made in 1839, when Schönlein first recognised the fungus of favus in man. Gruby in 1841 published a good description of this fungus, and Lebert in 1845 conferred on it the name of oidium Schönleinii. In 1842 Gruby discovered a trichophyton in sycosis of the chin, and in 1843 a microsporon in certain obstinate forms of ringworm.

The dermatomycoses of animals have long been known under the common name of "ringworm" or "tetter." In 1852 their parasitic character was suspected, as a result of the infection of men; in the following year Bazin discovered trichophyton tonsurans in a case of equine ringworm which had been transmitted to a gendarme. Some years later (1857) Gerlach published an important communication clearly proving the parasitic nature of ringworm in the ox. Almost simultaneously Reynal read a paper to the Paris Academy of Medicine in which he proved the transmission of ringworm from oxen and horses to man and the identity of these diseases with herpes tonsurans of man. From that time our knowledge of the dermatomycoses has become more precise and complete. The field steadily widens and the number of recognised parasites increases.

A reaction soon occurred. Even in 1850 Lowe had sought to prove that all the vegetable parasites which attack the human body were identical, particularly the achorion of favus and the trichophyton of tinea tonsurans; he regarded them as only lower forms of a single mould, viz., aspergillus glaucus. In 1859 Hogg expressed the same opinion, which was strongly upheld by Tilbury Fox and by Hebra, the great Viennese dermatologist. Hebra relied on the effects produced by applying to the human skin compresses covered with moulds. At that time Tulasne had discovered the polymorphism of certain fungi; his observations had been confirmed by Bary, Hoffmann, and Kühn, and it was natural to extend the facts to dermatophytes. Hallier was the warmest advocate of this generalisation of the doctrine of polymorphism. But the most eminent botanists have recognised that his experiments were marred by errors.

Somewhat later Grawitz revived these views, for a time with success, and regarded the fungi of tinea tonsurans, favus, pityriasis versicolor, and thrush as identical with oidium lactis. But all cultural experiments, and particularly those of Duclaux (1886), have shown the impossibility of transforming any dermatophyte whatever into oidium lactis or *vice versa*. Such experiments show Grawitz's mistake (which, moreover, was afterwards recognised) to have been due to variations in form of the dermatophytes under varying cultural conditions. At the present time clinical experience and mycology both agree in denying any community of action or nature between a dermatophyte and any mould whatever.

This does not mean that dermatophytes can only exist as parasites. Some of them may perhaps have a saprophytic origin, *i.e.*, may live on inert objects or on vegetables, developing and reproducing themselves there and infecting animals which accidentally become soiled with such objects. This is known to be the case with actinomyces and aspergilli, and we are not in a position to deny its possibility as regards dermatophytes.

Regarding the differentiation of species of the dermatophytes, a tendency has manifested itself among dermatologists during the past ten or fifteen years opposite to that which first followed the discovery of the parasitic fungi of ringworm. Cultural methods have led many experimenters to subdivide the former species, and to recognise almost as many forms as clinical varieties. The process has particularly been applied to trichophyton and achorion, and seems justified by the results of cultivation. But many of these new species have already been disallowed; it seems as if several of them only represent varieties of the main species, to which they revert after a sufficient number of cultivations. The formation of different pigments, on which the avowed differences are largely founded, does not seem sufficiently constant to serve as a criterion, and we have here only retained a few species whose authenticity has never been seriously questioned.

It may be mentioned that the principal genera—achorion, microsporum, and trichophyton—are linked together by intermediate forms which suggest their parentage.

The dermatophytes of the domestic animals belong to six distinct genera: trichophyton, eidamella, microsporon, achorion, lophophyton, and oospora, the essential peculiarities of which are shown by their cultures. Although cultures are necessary for the scientific determination of a parasitic fungus, they will not receive consideration in this paper, which deals more particularly with the characteristics as discovered by microscopic examination of the fungi *in situ*, in the lesions produced during their parasitic life.

(1) *Trichophyton* (Malmsten, 1848). *Mycelium* consisting of simple filaments or dichotomous ramifications with numerous septa, the divisions containing spores (mycelial spores); diameter 4 to 9 μ.

Sabouraud states that a specific distinction of trichophyta consists in the position of the fungus relative to the hair affected. The T. endothrix lives in the interior of the hair; the T. ectothrix outside the hair, which it surrounds like a sheath; the T. endo-ectothrix both within and around the hair. The T. faviformis of Bodin belongs to the third variety.

It does not seem that the division of the trichophyta of animals into endothrix and ectothrix is of real importance. The same trichophyton, inoculated on different species of animals, on similar animals of a single species, or even on different parts of a single individual, may indifferently present the endothrix or ectothrix appearances. Furthermore, in well prepared sections, the mass of the hair may seem invaded by the parasite, whilst single hairs when extracted and examined may show all the appearances of attack by a trichophyton of the ectothrix type (Dassonville).

(2) *Eidamella* (Matruchot and Dassonville, 1901). *Mycelium* extensively ramified, 1 μ 5 in diameter, with closely-placed septa, divided into approximately square or oval cells with one septum. (Cultures give perithecæ). One species only is known, affecting the dog.

(3) *Microsporum* (Gruby, 1843). *Mycelium* producing lateral ramifications, bearing conidia (conidial spores) 2 to 3 μ in diameter.

(4) *Lophophyton* (Matruchot and Dassonville, 1899). (Epidermophyton of Mégnin, 1881; not E. of Bazin, 1862, nor E. of Lang, 1879). *Mycelium* with certain filaments tortuous, thin-walled, usually without protoplasm, and sterile; others short, incurved, thick-walled, containing granular protoplasm (reproductive mycelium); no spores. Only one species known, affecting the fowl.

(5) *Achorion* (Remak, 1845). *Mycelium* with straight or curved filaments, from 2 to 3 μ in diameter, formed of cells about 12 to 15 μ long, with groups of two, three, or four branches, thick-walled, and capable of resolution into isolated, polyhedral cells (mycelial spores).

(6) *Oospora* (Wallroth, 1833). *Mycelium* with filaments 2 to 3 μ in diameter, intermingled with round or oval cells 5 to 6 μ in diameter, arranged in long irregular chains. In the dog (the parasitic species of the dog, O. canina, does not belong to the gymnoasceæ any more than the other oospora) the dermatophytes become attached to the epidermis, penetrate the hairs and epidermal cells, which they separate from one another and on which they live. The hairs fall away, more or less active irritation is set up, the animal shows a varying degree of pruritus, the bare patches appear reddened, and there is sometimes a certain degree of exudation.

The course of the disease is usually chronic, though it depends on the characters of the particular parasite, but in domestic mammals the prognosis is always favourable, and it is rarely difficult to destroy the parasite.

Ringworm results solely from contagion, and with the exception of age (young animals appear more susceptible to the disease) no general condition is known to affect its occurrence. Contagion may be direct, *i.e.*, from animal to animal, or mediate, *i.e.*, conveyed by tools, litter, etc. It also appears within the range of possibility that it may occur from a distance, by air-borne spores. Depending on the nature of the fungus, contagion may occur more or less easily between animals of the same species, between animals of different species, or between animals and men.

Vitality of the Dermatophytes.—Many experiments have been made to determine the period during which the spores retain the power of germination.

Gerlach collected trichophytic crusts during the autumn and preserved them in the laboratory, enclosed in paper capsules, until the following spring, testing them from time to time. Crusts preserved for more than three months proved ineffectual on the unprepared skin; but spores even as old as six months still germinated and produced characteristic lesions when applied to the skin after scarification. The only difference between the disease so produced and that brought about in the ordinary way was the longer period of incubation, the less density of the crusts, and the more rapid healing. Siedamgrotzky failed to transmit tinea tonsurans from the horse to the goat with crusts kept for five years and eight months. But he succeeded in transmitting the disease to the goat with crusts obtained from a bull eighteen months before. Mégnin also transmitted the disease to a dog with crusts preserved for eighteen months. Duclaux failed to obtain germination with cultures kept for two years.

Thin made a long series of experiments with cultures of trichophyton on gelatine; he found that after about two and a half years the spores lost all power of germinating, but he was successful with spores kept for eleven months. The spores of trichophyton contained in hairs were dead after eight days' immersion in water, but they resisted for two days. They also preserved the power of germinating after forty-eight hours' immersion in olive-oil, lard, or vaseline. One hour's contact with soft soap or 1 per cent. acetic acid destroyed them. Ten per cent. solution of carbonate of soda was less effectual: germination was still possible after three days. Sulphur ointments are of very moderate activity: spores resist for several hours. Citrine ointment is very active, destroying spores in one hour. Croton oil is absolutely useless, the trichophyton preserving its vitality after a week or more of immersion.

Diagnosis.—The diagnosis is based, primarily, on the most apparent of the clinical symptoms, but, however precise these may be, it is always advisable to make a microscopic examination of the affected epidermal scales. The parts selected are the newest, *i.e.*, the deepest layers of the crusts covering the circular patches and the hairs near the periphery of the patches. The hairs, etc., may be deprived of grease by immersion in absolute alcohol or æther.

By scraping the skin with a blunt scalpel one obtains specimens of epidermal scales and hairs. The abundant scurf covering the surface of the patches contains very few parasites, and specimens should be taken principally from the deepest parts. Hairs for examination should be plucked from the margin of the patch with forceps. It may be requisite to shave off a thin layer of the skin in order to discover spores which are absent in the crusts or hairs (Troester).

The material obtained is placed in a drop or two of 30 per cent. potash solution and carefully heated for a few seconds nearly to boiling point. By gently pressing on the cover-glass the cells, etc., are easily spread out for examination. To procure permanent preparations, the alkaline solution first used is replaced by glycerine, or, better still, by carbolic solution, which has not the disadvantage of rendering the objects too transparent, like glycerine. Sabouraud does not recommend the use of aniline stains, which produce opaque preparations difficult to examine: lactic blue is free from this disadvantage and can be used like potash solution. The preparation is viewed under a magnification of 500 to 800 diameters.

Without further precautions the observer will often see, at the base of one of the hairs, a whitish mass surrounding the root and formed of parasitic cells. If the hairs have already undergone extensive change the free ends may appear frayed out like a brush. One sometimes finds here and there between the crusts and the skin little masses of a dirty yellow colour, easily crushed and formed almost entirely of spores and filaments of the fungus. But it is only by studying all the products of growth that one obtains a complete idea of the nature of the parasite, and the specific characters are best made out by the method of culture.

The dermatophytes usually develop well on neutral or slightly alkaline media containing glucose. Sabouraud recommends the following formula:—

Neutral redistilled glycerine . . .	4 grammes
Distilled water	100 „
Peptone	1 „
Agar	1·50 „

One may also use agar prepared with sweet wort, potato, coagulated serum, peptonised bouillon, peptonised gelatine, or peptonised agar, but liquid media are less useful than solid for differentiating the various species.

Growth occurs even at 15° C.; at 33° C. it is most active; above 36° C. it is checked. The cultures are made in inclined tubes or in flat-bottomed flasks. It is often essential to grow certain species in small capsules at the ordinary temperature in order to observe certain conidia formations which are especially fragile. Free access of air is necessary, for the dermatophytes greedily absorb oxygen.

I.—RINGWORM IN THE HORSE AND ASS.

The various forms of ringworm which affect the horse have been ascribed to five species of dermatophytes, a microsporon and four trichophytons. To these must be added the verrucous trichophyton of the ass.

The different species comprise:—

(1) *Microsporum Audouini* (Gruby).—The mycelium found in the hairs assumes the form of axial bundles of filaments, 2 to 2·5μ in diameter, with well-marked septa. These filaments give off at right angles fine branches which pass towards the exterior, where they produce conidia (conidial spores), rounded or polyhedral in shape as a consequence of reciprocal pressure, brilliant in appearance, from

2 to 3 μ in diameter, and arranged without order around the hair, which they surround from its base up to a height of 3 mm. beyond the follicle. They render the hair greyish in colour and fragile.

M. Audouini var. *equinum* (Delacroix and Bodin).—This variety represents the form which the species assumes in the horse. It is distinguished from the type by the polymorphism of its cultures, which may assume three different forms.

(2) *Trichophyton mentagrophytes* (Ch. Robin).—This is a pyogenic trichophyton, producing white cultures (Sabouraud). The mycelium may be divided into chaplets of spores measuring 5 to 6 μ in diameter but of very irregular length, particularly towards the periphery of the hair (from 2 to 3 mm. up to 10 or 11 mm.). Endo-ectothrix.

(3) *Trichophyton flavum* (Bodin).—This trichophyton produces yellowish, wrinkled, vermicular cultures (Sabouraud). The mycelium may be divided into ovoid irregular cells from 5 to 6 μ in size; occasionally some may be found as large as 11 μ. Endo-ectothrix.

(4) *Trichophyton equinum* (Gedoelst).—This consists of an intrapilary mycelium composed of oval spores of from 4 to 6 μ in length and 2 to 4 μ in width. Surrounding the hair are ramifying mycelial filaments with numerous septa and few spores. Endo-ectothrix.

(5) *Trichophyton verrucosum* (Bodin) var. *equi.*—This is a faviform trichophyton producing brown, heaped-up, irregular cultures (Bodin). The mycelium rapidly breaks up into spherical spores with a double contour. Endo-ectothrix.

(6) *Trichophyton verrucosum* (Bodin) var. *asini.*—This is a faviform trichophyton producing grey cultures. The mycelium breaks up into regular chains of spherical cells. Endo-ectothrix.

Symptoms.—Two groups of ringworm affecting the horse can be distinguished, viz., the microsporic and the trichophytic.

(1) *Microsporic Ringworm.*—The commonest form of this disease is the condition known under the name of contagious herpes in foals. It almost exclusively attacks young animals. It is indicated by the occurrence of bare spots, usually circular in shape, which principally attack the upper parts of the body (shoulders, neck, sides, and head), but often the croup and back. They are rare under the belly and on the limbs, and are never seen at the base of the poll, mane, or tail. The period of incubation varies between eight and twenty-four days.

At first, very small vesicles may be present, but they dry up in twenty-four hours. Even under experimental conditions and where the development of the disease can be carefully watched some care is needed to detect them. The abundance of hair and the pigmentation of the skin usually prevent the primary signs of eruption being discovered.

As a rule the first signs observed are circular patches about the size of a shilling, sharply defined against the uninjured coat, and due to the dry appearance and vertical position of the hairs. The hair falls away in a few days, and the bare spot which remains is often the first thing to attract attention. Falling of the hair is hastened by grooming.

The hairs at the periphery of the spot are first shed, the lesion thus assuming the form of a ring. Although this appearance closely resembles that seen in herpes circina of man it is due to an entirely different cause. This stage only lasts for a short time; very soon the whole surface of the spot becomes bare, and, if the disease is extending, the animals coat assumes a characteristic appearance, being dotted over with circular bare spots.

The hairs are not expelled from the follicles but break off almost level with the epidermis, their broken extremities being irregular and resembling a worn paint brush.

The epidermis of the patch falls simultaneously with the hair; it appears to undergo softening and the skin assumes a blackish-grey tint and becomes slightly moist, a fact which has been attributed to rupture of vesicles, though no actual proof has been given. The moistness soon disappears, however, the surface drying and becoming covered with epidermal scales of varying thickness, agglutinated into flat crusts which are continually shed and renewed. These crusts often have a greyish or yellowish appearance, resembling asbestos.

At the same time the lesions extend peripherally, sometimes attaining the diameter of a five shilling piece. Each zone as invaded exhibits the above described appearances. Having attained this size the spot ceases to grow and the crusts to form. A smooth, dry, dull, slaty-grey surface remains, from which branny scales are shed. Gradually the hair grows again, though it exhibits a deeper tint than the rest of the coat, except in the case of light grey horses.

Recovery is only local. Other spots form in the vicinity of the original ones and go through similar phases. Spots may also become confluent, forming more or less extensive diseased surfaces, which are very difficult to cure.

Fleming has described a circinal variety which may be compared to trichophytic circina in man both as regards its form and method of spread, it being due simply to rapid healing of the central portions of the spot whilst the disease continues to extend towards the periphery. The lesion then assumes the form of a more or less irregular ring.

In the microsporic form there is very little itching, though slight rubbing seems to cause the animal pleasure.

One attack does not prevent a second. Thus ringworm may again appear on the site of an old (healed) patch (Fröhner).

I found great numbers of a microsporon in body-dust and crusts taken by Cousin from a Guadeloupe mule, the skin, and particularly the limbs, of which were almost devoid of hair. The hair had only begun to fall away some two months before.

A form of ringworm presenting clinical features quite different from the preceding was seen by Le Calvé and Malherbe. They obtained a parasite which Bodin recognised as specifically identical with the microsporon of herpes in foals, but differing from it by its power of directly producing in cultures the oospora form, whilst in the type this form proceeds from the endoconidium and acladium forms.

This type of microsporosis is indicated by bare patches, which appear for several years running, recurring in spring, but readily disappearing after simple treatment. The patches are numerous, rounded in shape, very sharply marginated, of regular contour, and measure from 7 to 20 mm. in diameter; they are generally smooth, often confluent, and do not produce proliferation of the epidermis or pruritis. They may affect any part of the body, but are commonest on the limbs. On parts untouched by the curry-comb their surface is covered with greyish crusts which are only slightly adherent and include the broken shafts of the hairs. The hairs are loose and broken off level with the skin. Towards the periphery the hairs are still whole, but are thinner and contorted though still fairly adherent. Along their length, and particularly near their bases, can be seen yellowish masses, which under the microscope prove to be formed of spores of the microsporon.

(2) *Trichophyton disease.*—(a) *Trichophyton flavum.* The lesions consist of patches from 8 to 10 cm. in diameter, more or less regular in shape, greyish, and completely denuded of hair. The hairs are lifted and glued together at their base by a yellowish-grey crust along with which they soon fall. The bare patches do not extend or exhibit any trace of suppurative folliculitis (Bodin).

(b) *Trichophyton equinum.*—In this form the patches are numerous. Some are isolated, and are distributed over the croup and shoulders; they attain a diameter of 3 centimetres or more. Others are found near the posterior margin of the saddle bed; they are often confluent, and at times cover a space 5 to 20 centimetres in diameter. At first they are only perceptible to the touch, but as the hairs are rapidly shed they soon become visible. The least traction or friction displaces a squamous crust carrying with it a mass of the affected hairs. The skin then appears smooth, moist, reddish, or light grey in colour. The spot soon dries, becomes covered with branny or mealy crusts, and assumes a slaty-grey colour. The lesions grow in size by extending peripherally (Matruchot and Dassonville).

(c) *Trichophyton verrucosum* var. *equi.*—The spots usually attain 5 to 6 centimetres in diameter, are numerous, and affect the shoulders, neck, flanks, croup, or whatever the harness touches. They may become confluent, and thus produce extensive patches with irregular margins. At first the hairs are raised but not broken, and are glued together at their base by a greyish, soft crust. In less than a week this crust falls, carrying with it the hair and leaving an absolutely smooth grey surface, covered with dry, greyish, more or less abundant crusts; there is no suppuration of the hair follicles. In young horses, however, removal of the crusts exposes vesico-pustules, and the surface of the patch is seen to be slightly raised, reddish, and indurated (Bodin).

(d) *Trichophyton verrucosum* var. *asini.*—The lesions were similar to those of the preceding form and of trichophyton disease of the horse, but in the case observed were confined to the neck, head, and ears (Bodin).

Trichophyton disease in the ass has been described by Galtier and Mathis.

(e) *Trichophyton mentagrophytes*—This form of trichophyton disease usually attacks the nostrils or head. It forms patches, sometimes 5 to 6 centimetres in diameter, resembling in appearance the pustules of horse-pox in process of healing. The bases of the hairs covering the whole patch are glued together with a brownish soft crust of unequal thickness. By slight traction on the hair the crust can be removed, exposing a prominent red fungating surface dotted over with minute depressions exhibiting greyish bases. These represent the openings of minute pustules, some of which may generally be found near the margin of the spot. The hairs are expelled but are not broken. The disease, therefore, is of the nature of suppurative folliculitis. It develops rapidly, and, as a rule, recovery occurs in from four to five weeks.

This is the form described by Mégnin. It most closely resembles trichophyton disease in bovines. The crusts are yellowish, not grey. The patches differ from those of microsporic disease in being absolutely smooth. This form of trichophyton disease has frequently been seen. It may in certain cases spread with extreme rapidity; a horse, apparently healthy the day before, was found covered with more than five hundred patches (Evrard). A case of skin disease encountered by Weber was ascribed by Mégnin to trichophyton mentagrophytes. The animal showed papules about a centimetre in diameter covered with a slightly adherent crust which, when removed, left exposed a bare, dark grey, slightly granulating surface resembling the cicatrix of a variola pustule.

Course, Duration, Termination, and Prognosis.—As a general rule a patch of ringworm never exceeds a certain maximum diameter, and when this has been attained spontaneous recovery follows, the fungus having exhausted its local activity. Rubbing may, however, transfer spores to other points, and lead to the formation of fresh patches which pursue a similar course. If these are close to the original they may unite with them, and thus form the large surfaces which have so frequently been described. Even this method of spread gradually ceases, however, and the animal eventually recovers.

A local patch usually remains in existence for forty to fifty days. The disease is much more obstinate in young animals than in adults. Want of cleanliness facilitates the increase of spots and postpones recovery indefinitely. Animals with fine skins, or animals which have been clipped, are well nourished, and well looked after, recover much more rapidly than those under the opposite conditions. Shedding of the coat favours recovery, a fact which explains the influence of the advent of spring and of grazing.

It is scarcely probable that issues or rowels have the favourable influence which Reynal believed. They may possibly have been more useful in strangles (in which he also advised their use), but this also requires confirmation.

The disease seldom proves very rebellious to treatment, contrary to what occurs in ringworm in man. As the disease dies out the hair again grows, and the spots can at once be distinguished from those where the process is still active. The skin no longer exhibits crusts or scales, and, although the hairs remain thinner and darker in colour for a time, they are uninjured and are not broken or frayed at their extremities.

Ringworm in the horse is a benign disease, which does not disturb the general health, seldom proves obstinate to treatment, and may even undergo spontaneous recovery. The general symptoms formerly ascribed to it were the result of pathological conditions which had prepared the way for the parasite.

The gravity of ringworm depends entirely on its contagious character. It may extend to all the animals on a farm, in a stable or regiment; this fact is chiefly important on account of the malignant character it assumes when it attacks stablemen, etc.

Diagnosis.—It is difficult and sometimes impossible to differentiate the different forms of ringworm in the horse by clinical examination. That due to trichophyton mentagrophytes is readily distinguished by the signs of suppurative folliculitis which accompany it. The others, however, all produce circular spots covered with a crust which glues together the bases of the hairs and are later succeeded by smooth bare patches. The diagnosis of the parasite can only be arrived at from cultures carried out by a specialist. Furthermore, it is possible that later investigations may modify our views regarding several species of trichophyton in the horse.

The parasitic nature of ringworm always enables us, after microscopic examination, to separate it from the other dermatoses which clinically resemble it. The round form of the patches, the appearance of the surfaces, the condition of the hair, the contagious character of the disease, and the various points more fully set forth in the foregoing pages all point in one direction.

Psoriasis circina of the horse, described by Mégnin, appears to be a herpetic disease in which no parasite has hitherto been discovered. The hairs are shed without undergoing change. The disease resists all forms of anti-parasitic treatment and yet yields rapidly to the administration of iodide of potassium.

The form of herpes associated with strangles produces round or oval patches, but the hairs are glued together by a purulent secretion. They fall away in three to four days and are rapidly replaced by a new growth of fine and abundant hair.

Causation.—The essential cause of any form of ringworm is the parasite, but certain conditions favour development and contagion.

Weak, unhealthy, and exhausted animals appear more subject to the disease. Want of cleanliness and general care favour the implantation of the fungus, facts which usually explain the greater frequency of ringworm in certain countries. Youth has a real predisposing influence; even sucking foals are not exempt, and young animals are particularly liable to the disease, as proved by experience in the army. Yearlings are more frequent sufferers than two-year-olds and three-year-olds, and in them the patches develop more rapidly. Black, bay, brown, chestnut, and cream-coloured animals are more readily affected than grey or white. The same is true of thoroughbred as compared with half-bred foals (Fröhner). Remounts drawn from Normandy contain an excessively large number of subjects. Ringworm rarely attacks horses in Australia, though in oxen it is common (Fleming).

The disease sometimes assumes the epizöotic form. Thus in 1840 it attacked several hundreds of horses in the valley of Borne in Savoy (Papa). Regiments of cavalry sometimes suffer in a similar way.

Contagion.—(1) *From Horse to Horse.* The occurrence of epizootics of ringworm in horses would in itself have aroused suspicion of the contagious nature of the disease, but the earliest exact record appears to be that of Bouley the younger in 1852. A young horse suffering from a ringworm lesion arrived at the remount depôt at Caen, and communicated the disease firstly to its immediate neighbour, and afterwards to seven other horses kept in the same stable. Two of the latter, sent to the veterinary college at Alfort, transmitted the disease to two other horses standing next them. Contagion was effected by curry-combs, brushes, bits, and rings which had been used on the diseased animals.

This form of contagion is common in regiments. It may result from wearing infected harness or saddles. Mégnin in 1882 described a case in which a saddle played a remarkable part. Nearly 200 horses in his regiment were successively attacked with ringworm, all of the early cases showing patches on the left side of the saddlebed. This was due to the fact that the first horse had a lesion in this position, and the saddle having been used successively on several horses had inoculated each one in the same position with the spores of the fungus. Many other cases have been noted in cavalry regiments. Finally, clipping machines and scissors may play an important part in conveying the disease.

(2) *From Animals other than the Horse to the Horse, and vice versa.* Gerlach succeeded in transmitting ringworm from the ox to the horse: "at the end of eight to ten days the signs of contagion were not fully defined; a circular spot with sharply defined margins appeared during the third week; towards the fourth or fifth it had disappeared, leaving behind a bare, flexible patch. The crust was thinner than in bovine animals and contained very few fungi. The hair did not break."

Mégnin in 1878 was also successful in transmitting ringworm from the calf to the horse.

Reynal in 1852 conveyed ringworm from a horse to two calves by cleaning the latter with curry-combs, brushes, and wisps which had been employed on two infected horses. Nettleship made similar observations.

Siedamgrotzky inoculated a dog, two sheep, and two pigs from a horse.

The ease with which ringworm in the horse can be conveyed to the ox and *vice versa* is due to the fact that one of the most common forms of ringworm in the horse, produced by trichophyton mentagrophytes, is also the common form in the ox.

The microsporon of the horse is readily conveyed to the guinea-pig (Bodin) and to the dog (Le Calvé and Malherbe). It is also met with in one of the forms of ringworm in carnivora.

(3) *From the Horse to Man.*—According to Zürn the transmission of ringworm from the horse to man was first observed by Papa in 1848. Papa's cases occurred during an epizöotic of ringworm which attacked horses in the valley of Borne in Savoy, but the first detailed observation is due to Bouley the younger and Reynal in 1852, and refers to the horses mentioned in former pages. The gendarmes who cleaned these animals contracted the same disease, and one of them communicated it to his wife and daughter. The two horses which were sent to Alfort also infected the pupil and the groom who looked after them. Bazin regarded these attacks as of the nature of herpes circina. Some years later Galligo published a new case of conveyance of the disease from a horse to its driver.

After 1871 reported cases became more frequent. Tilbury Fox at that time communicated to the Clinical Society of London a case of transmission of herpes circina or ringworm from a pony to seven human beings. Fleming, Tilbury Fox, and Horand reported further cases in 1876. Dieu reported an epidemic of herpes circina transmitted from horses to the soldiers of a regiment. In eight months twenty-two cavalrymen were successively infected by young horses which had recently arrived at the remount depot of Caen. Larger saw nearly one fifth of the men in a regiment of dragoons attacked with herpes circina of equine origin. Similar facts are mentioned by Mégnin, Aurregio and Touvé, by Gerlier and lastly by Longuet, who gave an excellent resumé of the facts.

Considering the frequency of ringworm in the horse, contagion to man is very exceptional, for there is scarcely a regiment in which some young horse does not exhibit the disease.

Grooming is the most ordinary method of contagion, and almost all the observed cases have been referred to it; one, however, was referred to rugs removed from horses and used by men (Mégnin).

The Ferney-Voltare epidemic of ringworm described by Gerlier first attacked the children of a horse-clipper whose hair the father had clipped with his machine.

The rubbing which accompanies grooming facilitates inoculation. The principal seat of eruption is the forearm and wrist, portions of which are usually uncovered during the work. In Mégnin's cases the men who used rugs from horses suffering from ringworm were attacked about the face and neck. The period of incubation varies between five and ten days.

Trichophyton disease attacks by preference parts free of hair, and seems, like that of bovine origin, a form of tinea circina; cases of equine origin exhibit special intensity; often, in fact, they are as severe as those derived from the ox. Gerlier states that the trichophyton of the ox causes true inflammatory sycosis, producing boils and even anthrax (?) Spontaneous recovery is said to be more frequent, but is followed by the formation of cicatrices; in children the parasite is said to be incapable of producing trichophyton disease in parts covered by long hair. In this respect it differs from the trichophyton of the horse, which has a greater tendency to attack man, to affect the epidermis, and to be more obstinate to treatment.

These conclusions were arrived at before the dermatophytes of the horse were differentiated. We now know that the various species are unequally contagious for man and produce quite distinctly differing lesions.

The microsporon of the horse produces on smooth portions of the skin little erythematous fugitive lesions resembling the circina form of trichophyton disease at its period of full development. The lesions heal spontaneously or yield to very simple treatment (Bodin). It is stated, however, that in children this parasite of the horse produces bare spots in places usually covered with hair (Bosellini).

During an epidemic of ringworm produced by trichophyton equinum several of the men who had charge of the horses showed spots, usually situated around the neck. Experimental inoculation on the forearm of a man produced a similar spot (Matruchot and Dassonville).

In man, as in the horse, the trichophyton mentagrophytes produces an expulsive suppurative folliculitis. On portions of the skin destitute of hair the points are closely crowded together. In the chin region the disease resembles sycosis, and in parts normally covered with hair assumes the character of kerion celse. These diseases are not very difficult to treat.

The trichophyton flavum, which is very contagious for man, produces in his case lesions similar to those of the horse, viz., a simple epidermitis with more or less extensive spots, slightly moist on the surface, and not of a very obstinate character (Sabouraud.)

The trichophyton verrucosum var. equi., which is also very contagious for man, produces an expulsive follicular suppuration. The affected area projects beyond the general surface, assuming a slightly domed shape (Bodin).

The trichophyton verrucosum of the ass is also readily conveyed to man. It produces suppurative folliculitis, the spots being raised very distinctly above the surrounding skin, as in kerion celse. The surfaces are rough, project somewhat, and exhibit a few whitish vesico-pustules.

W. Williams described a case of "tinea favosa" transmitted to three horses and about twenty oxen occupying the same stable by cats which suffered from ringworm sleeping on the animals' backs. This case was undoubtedly caused by the trichophyton felineum of the cat.

Treatment.—Preventive treatment consists in careful attention to cleanliness and regular grooming. The stables occupied by horses suffering from ringworm, the tools used for cleaning them, and the harness they wear should be rigorously disinfected. Tools, etc., must never be used on healthy subjects after having been employed for those suffering from ringworm. When ringworm is limited to a small area its extension can be prevented if the stableman will avoid touching the diseased spots with tools used for cleaning, and so carrying the spores of the fungus to other points.

Mégnin declares that he radically cured several young horses by simply scraping the crusts, cautiously removing all loose material and broken hairs, and at once burning them.

It is usually advisable to clip the animals before commencing treatment. All cleaning should be done out of doors.

It is impossible to prevent the loss of hair from a spot in course of development. The object of treatment is to prevent the disease extending by destroying the parasite in the hairs and crusts.

Beel has shown that washing favours extension of the disease by softening the epidermis and hair.

As a general rule, ointments and oily preparations are more useful than alcoholic and aqueous preparations.

Almost all the materials commonly employed give good results, for in domestic animals the growth of the parasite is easily checked or stopped by medical applications. Simple materials are best. Irritants should be avoided, as they are apt to leave lasting and even indelible marks.

The applications most commonly employed are solution of corrosive sublimate in dilute alcohol (1 in 300), carbolised glycerine or oil (1 in 10), tincture of balsam of Peru, pure alcohol, petroleum, oil of cade, ointment of white precipitate (1 in 4), ointment of red precipitate (1 in 8), ægyptiac, tincture of iodine, or an ointment composed of one part of carbolic acid, five parts of lard, and five parts of soft soap. Thin recommends pure soft soap used as an ointment.

The applications are repeated once or twice a day or every second day, according to the nature of the material chosen and to its local irritant action.

Faurie and Le Calvé recommended Cutler's formula, composed of crystallised carbolic acid, tincture of iodine, and chloral hydrate in equal parts.

The hair is clipped away, the parts washed with soap and dabbed with the fluid, taking care to extend a little farther than the visible margin of the diseased spot.

Recovery is indicated by the appearance on the spot of a crop of fine, closely-placed hairs.

(*To be continued*).

NOTE ON THE METHODS OF COMBATING RINDERPEST.

By STEWART STOCKMAN, C.V.O., Board of Agriculture and Fisheries,
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SINCE May, 1903, fourteen outbreaks of rinderpest have been dealt with in the Transvaal. Most of these occurred in or near native locations, where the disease had probably been lurking since the close of the war. It is rather characteristic of this disease, if we may judge from its history in Western Europe before the introduction of inoculation, that a first outbreak sweeps a newly invaded country almost from end to end with very fatal results. It then seems to lose its epizoötic characters and assumes those of an enzoötic; that is to say, it breaks out every now and again in certain centres, from which it does not tend greatly to spread. There are several reasons for this. In the first outbreak most of the more susceptible individuals are killed out, while a certain percentage pass through the disease and acquire immunity. The result is a more or less immune community, which deprives the disease of its epizoötic qualities. To this one must add that the owners of stock, warned by past experience, are less inclined to risk their animals by taking them into the vicinity of an infected area. For a considerable time, however, a certain number of susceptible individuals continue to exist, and these serve to keep up the infection in this or that locality. In the East one has a good opportunity of studying this phase of rinderpest. The disease has been established there for centuries, practically without interference except that which arises from natural laws. The result is that the indigenous cattle by a weeding-out process have acquired a high degree of hereditary resistance which is not yet absolute; only the less susceptible animals can survive in this zone of permanent infection, and, naturally, they are the only ones which propagate their species and characteristics. A certain number of the progeny, however, are without doubt born less resistant than others. Should the disease die out for a time in one part of a province, as it sometimes does, the more susceptible individuals increase largely in number, and on the next visitation of the plague the death rate is unusually high. This one can see in certain of the extensive provinces of India. For example, Madras was estimated some three years ago to contain about

10,000,000 cattle and buffaloes, in spite of rinderpest. At the time of the writer's service in that province small outbreaks of rinderpest were being reported in various parts, but the greater portion was then free, probably because severe outbreaks had occurred in previous years. One can easily understand, however, how, owing to the movement of transport cattle, the disease could come out from its lurking places as soon as the animal soil is favourable to it. Our position in the Transvaal after the signing of peace was rather similar to the above. That is to say, the cattle of the country, which had recently been swept by the disease, were as a whole fairly resistant to rinderpest, but some 40,000 green cattle had been imported for repatriation purposes, and it was highly important that infection should be kept away from these cattle. Moreover, the free use by laymen of the various methods of inoculation had produced quite a number of partially immune animals, while others stated to be salted were not resistant at all. The position, then, was a rather serious one, and it was further complicated by a dangerous trade, that of the so-called "private inoculator," having been allowed to establish itself. The private inoculator traded chiefly with the natives, whom he charged for inoculation at the rate of £1 per animal. He used principally the virulent blood method, which is well known as a means of starting fresh centres of infection, and he moved ahead of the disease, trying to persuade natives to have their cattle inoculated.

By Government Notice No. 834, 42 (*h*), of 1903, it was made illegal to inoculate with virulent blood or bile, except by permission of the Commissioner of Lands.

Up till May, 1903, the prevailing custom was to employ the virulent blood method. It just so happened that on account of the large number of highly resistant animals in the locations this practice did not result in an extensive dissemination of infection. It rapidly killed out the more susceptible animals, and in a short time produced a highly immune remnant. It could not be allowed to go on, however, as, in addition to being a destructive method, it was calculated to bring the disease nearer and nearer to the green herds. The "serum alone" method was then adopted exclusively. For the successful employment of this method, which does not disseminate infection, I think the following points must be considered :—

(*a*) That the virulent material does not remain active for more than a day or two outside the animal body. In the plains of India blood from a sick ox could not be depended upon to keep its virulence for more than forty-eight hours, even when kept in the ice-chest.[1]

(*b*) That it does not remain active inside the living organism for a longer period than fourteen days, except in what are known as chronic bowel cases, but thirty days is a safe limit at which to put to its activity in the latter.

(*c*) That the disease smoulders on in a densely stocked location owing to the occurrence of mild cases amongst the partially resistant animals, which pass unobserved. These and the chronic cases mentioned in (*b*) may start a fresh outbreak if they be brought in contact with suitable fuel.

(*d*) That, as the disease is to be attacked by rendering the animal organism unsuitable for the upkeep of virus, the resistance conferred must remain as an absolute immunity for a period in excess of the longest possible duration of activity on the part of that virus—under thirty days in the chronic cases—if the disease is to be eradicated from a herd.

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[1] The experience in South Africa was the same.

(*e*) That serum alone cannot be depended upon to give absolute immunity for a much longer period than ten days, therefore the injections must be repeated three times with that interval in order to protect an animal for a month (the outside limit of infection).[1]

(*f*) To ensure success, the serum must be injected to all the cattle likely to come in contact with the infected; it will of course save serum if the actually infected herd can be properly isolated from its neighbours.

The outbreak in the Gopani Native Reserve was the last we had to deal with, and so instructive was its history that I propose to give some of the more interesting details. Rinderpest was reported at Gopani on the 17th April 1903. I purposely say reported, because no infecting animals could be traced, and it is assumed that the disease had been quietly smouldering for some time. The part where the disease broke out is known as Schulputs Valley, which contained herds belonging to different owners, and the total number of animals was about 800. Although all the cattle did not belong to the same owners, this is to be regarded really as one outbreak. It lasted in the various herds concerned from April to 13th September 1903, when the last death occurred. It was conspicuous in the later stages by the number of recoveries and the low death rate, which proves that we had to deal with highly resistant cattle. The mortality varied from 58 per cent. to 1·6 per cent. in the recurrent outbreak.

In the first affected herd glycerinated bile was used, as no serum was on the spot. The first death was reported on 17th April, and the last occurred on 3rd May. Mortality 58 per cent.

The second herd became infected on 21st May. Owing to verbal instructions received through the South African Constabulary Department, which was previously to May supervising outbreaks of contagious disease, this herd was in error treated by the virulent blood and serum method. This last death occurred on 12th June. Mortality 12 per cent.

The third herd was reported sick on 20th August. Instructions had in the meantime been issued to employ serum alone, to use it on all the cattle which might possibly come in contact with the diseased, and to inject it every ten days. The last death occurred on 13th September. Mortality 10 per cent.

The fourth herd became sick on 30th August—that is to say, practically at the same time as herd three, and not after a full month's interval, as in the other cases. It may be assumed, I think, that this was one of the last uninterrupted outbreaks.

It is exceedingly interesting to note that the owner of this herd, Mailaglis, refused to have his cattle inoculated before the disease broke out. otherwise they would have been included from the first amongst those receiving periodical doses of serum. The herd was treated in the same way as the third herd The isolation precautions, however, were more complete. The last death occurred on the 30th August. Mortality 1·6 per cent. The District Veterinary Surgeon was practically living in the location at the time, and he injected serum to all the animals the day after the outbreak was reported.

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[1] This was my experience in India. I am aware that other experimenters, after using large doses of serum, have credited it with giving a slightly longer period of absolute immunity than ten days. It is safer, however, to accept the shorter period.

The doses of serum employed in the above cases was from 50-200 cc.

Although the disease had apparently ceased by the 13th September, it was resolved to take no risks regarding the possibility of mild cases being still in existence. To make certain, four green oxen were purchased and imported into the reserve on the 29th September, sixteen days after the last death. They were allowed to run with the other cattle in Schulputs Valley for about a month. As they remained healthy it was considered that rinderpest had been stamped out, but to avoid all risk quarantine was kept on until the 3rd December 1903, when the Transvaal was declared free of rinderpest. No other case has occurred since.

The manufacture of serum has now ceased, but a stock large enough to deal with initial outbreaks has been kept on hand. Should the disease ever come in again, which is possible, I do not think it need be allowed to run through the country. I advised that no other inoculation than the "serum alone" method be permitted where it is possible to carry it out. The Veterinary Conference, held in Bloemfontein in December 1903, passed the following resolution on the subject of eradicating rinderpest:—

"The means best suited to attain this end are the stamping out of outbreaks of rinderpest by a liberal use of serum if obtainable; failing this, pure bile inoculation, carried out under professional supervision, is preferable to any other method."

In conclusion I should like to add a few remarks relative to the dose of serum to be used by the "serum alone" method. As the best method of cutting short an outbreak and preventing its further spread, its adoption is only justifiable if it be used in such a manner as will insure absolute immunity to the "in contact" animals for a time equal to the duration of infection. Even amongst herds which have been for some time in what might be called a stricken country the immunity of the individuals varies to a considerable degree, and one must legislate, so to speak, for the least immune. As one has no means of estimating the degree of immunity possessed by each individual, one must fix the dose—a large one—in relation to the most susceptible. In doing so, it must not be forgotten that anti-rinderpest serum is not prepared with a fixed virus, nor is its strength constant. If one neglects these considerations, and tries to be too economical with the serum, it happens that some of the more susceptible take rinderpest, and they infect the others as the effects of the serum are passing off. The disease may thus be kept smouldering on for an indefinite period. It is admitted that the curative effects of serum are not great. The test, then, of efficacy of serum when used on more or less immune herds is not so much to be found in the number of recoveries, which may have very little bearing on the subject, as in the disappearance of infection.

THE ARTIFICIAL IMMUNISATION OF CATTLE AGAINST TUBERCULOSIS.[1]

By LEONARD PEARSON and S. H. GILLILAND.

(From the Laboratory of the State Live Stock Sanitary Board of Pennsylvania.)

A Paper presented to the 42nd Annual Meeting of the Veterinary Medical Association, at Cleveland, Ohio, 15th to 18th August 1905.

EFFORTS to produce artificial immunity against tuberculosis have been conducted for a number of years and along many lines. In 1890 Robert Koch announced the discovery of tuberculin. It was claimed at that time that by the use of tuberculin resistance to tuberculosis could be increased in such a way as to assist in withstanding infection, and also that some infections already established might under the use of tuberculin be overcome. But extensive experimentation has shown that these effects are not sufficiently uniform, lasting, or powerful to be of distinct practical value.

Following this pioneer work of Koch came the work of many investigators with modified tuberculins and with extracts from tubercle bacilli made in a variety of ways. Repeated experiments with all these substances have shown that it has not been possible by their use to render animals immune to tuberculosis permanently, or to a practical degree. Experiments have been made by several investigators to test the immunising value of dead tubercle bacilli and of bacillary pulp. The results have been about the same as have followed the use of extracts from tubercle bacilli.

It became evident years ago that immunity against tuberculosis, to be of value, must confer protection against the organism of tuberculosis as well as against its toxins. There must be bacterial as well as toxic immunity. Efforts to immunise animals against living and virulent tubercle bacilli by inoculating them with living tubercle bacilli of low virulence were made as early as 1891 by Granchez and Ledoux-Lebard. In 1892 and 1893 Trudeau[2] found that by inoculating rabbits subcutaneously with living cultures of avian tubercle bacilli, he was able to increase their resistance to infection from mammalian tubercle bacilli of a culture known to be virulent for rabbits. De Schweinitz[3] in 1894 discovered that it was possible to very greatly increase the resistance of guinea-pigs to inoculations of tuberculosis by inoculating them with attenuated tubercle bacilli of human origin ; the process of attenuation consisting in prolonged cultivation (twenty generations) of glycerine beef broth of acid reaction. Tubercle bacilli of human origin grown in this way lost their virulence for guinea-pigs, but guinea-pigs inoculated with this non-virulent culture developed so much resistance to tuberculosis that when they were afterwards inoculated with tuberculous tissue from a cow they remained healthy, while other guinea-pigs inoculated with the same material from the cow died of tuberculosis in seven weeks. De Schweinitz injected very large quantities of human tubercle bacilli into cattle subcutaneously, intravenously, and intraperitoneally. He found that by gradually increasing the dosage enormous quantities could be tolerated without injury. He administered as much as 500 cc. of a heavy suspension of tubercle bacilli at one time.

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[1] Reprinted from the "American Veterinary Review," September 1905.

[2] "New York Medical Journal," 23rd July 1893.

[3] "Medical News," New York, 8th December 1894.

M'Fadyean[1] in 1901 and 1902 reported that he had found that the resistance of cattle to tuberculosis may be very greatly increased by the use of successive inoculations with tuberculous material or tubercle cultures of low virulence. In a paper of M'Fadyean's entitled "Further Experiments Regarding the Immunisation of Cattle Against Tuberculosis,"[2] the author states this conclusion :—

"It appears to be justifiable to conclude that, whatever may have been the degree of natural immunity possessed by these three experimental animals, it was much increased by the successive intravenous inoculations to which they were subjected. The immunity was not absolute, but it may be doubted whether a degree of resistance that would merit that term is obtainable by any method in cattle."

Von Behring announced in December in 1901 that he was engaged in studying the immunisation of cattle against tuberculosis, and he has since issued several reports upon his work. Von Behring has made a very large number of experiments in this field; he has endeavoured to produce immunity by the use of tuberculins, by the use of other tuberculosis toxins, by anti-toxins, by the use of dead tubercle bacilli, of tubercle bacilli weakened by chemical agents, and by the use of tubercle cultures of low virulence. A method for the vaccination of cattle against tuberculosis has been formulated by von Behring, and it is based on the use of tubercle bacilli of human origin that are non-virulent for cattle. In making this "vaccine" the tubercle bacilli are dried and ground to a powder. The vaccine material is sent out in this dried state. It is necessary to make a suspension of it in normal salt solution, whereupon it may be administered by intravenous injection. A number of investigators, as Lorenz, Schlegel, Eber, and Hutyra, have tested the resistance of cattle treated by von Behring or according to von Behring's method. It has been shown that the resistance of these animals to tuberculosis has, in most cases, been increased, and sometimes considerably so.

It is to be observed, however, that many of the cattle reported upon by the above authors were not vaccinated according to the method that von Behring now recommends, and as Eber says[3] ". . . The tuberculin test is not a reliable means for determining the freedom from tuberculosis of an animal that has been treated with mild bovine or human tubercle bacilli unless a long time (generally more than half a year) has elapsed since the last administration of infectious material. . . . Since neither of the treated cattle was vaccinated by the two vaccination methods with attenuated human tubercle bacilli according to the method now recommended by von Behring, the conclusions that are reached as a result of my investigations have only a relative bearing on an estimation of the value of the method now recommended by von Behring.

"The results of the investigations show, however, that it is possible to confer upon cattle by treating them with attenuated bovine or human tubercle bacilli a certain degree of resistance to artificial tuberculosis infection.

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[1] "Journal of Comparative Pathology and Therapeutics," June 1901, and March 1902.

[2] "Journal of Comparative Pathology and Therapeutics," March 1902.

[3] "Zeitschrift fur Thiermedicin," Band IX. Heft 3-4, 1905.

"Whether this resistance, as produced by the two-vaccination method, with attenuated human tubercle bacilli, that is now in use, furnishes protection against natural infection can only be determined by years of careful observation on the greatest possible number of animals immunised in this way and controls at the time of slaughter."

Hutyra[1] has reported some work conducted by him in 1903 and 1904 at the Royal Veterinary College of Hungary, wherein he tested the immunity of calves vaccinated according to von Behring's method, with material furnished by von Behring, and also with material prepared from cultures made by himself. Hutyra tested the resistance of his vaccinated animals by inoculating them with virulent bovine tubercle bacilli, and their resistance was compared to that of unvaccinated animals. He found that nearly all of the vaccinated animals had more resistance than the unvaccinated animals. One animal treated with von Behring's vaccine (No. 3) appeared to have received no immunity as a result of vaccination. Hutyra's own vaccine material from different sources appeared to give a noticeably higher degree of immunity than that obtained from von Behring's.

Recently, a large number of cattle, amounting to several thousands, have been vaccinated after the von Behring method in Germany, Austria, and Hungary. The process does not appear to be free from danger in every instance, as is shown by the reports of Marks, Casper, and others. It is as yet impossible to draw any conclusions whatever as to the efficacy of vaccinations from the results of this great number of vaccinations of cattle on farms. The only conclusion at which one may fairly arrive is that vaccination appears, in most cases, to be unattended by danger to the vaccinated animals. Whether immunity is conferred and, if so, whether it is sufficiently powerful or lasting for practical purposes, is not yet shown by the practical application of von Behring's method. Of the animals that have been vaccinated on farms and afterwards exposed to infection a number have been slaughtered, and some have been found to be afflicted with tuberculosis, while others have been found to be free from tuberculosis. The presence of lesions of tuberculosis is taken by some of von Behring's observers to mean that the animal was tubercular before it was vaccinated. Of course, this may be true. But it must be remembered that the absence of lesions of tuberculosis in vaccinated animals cannot fairly be taken to signify the existence of a serviceable degree of immunity, unless it is shown that the conditions under which these animals were kept were such as to lead to the infection of a considerable proportion of unvaccinated animals. And this evidence is in most cases lacking.

Klimmer has carried out some very useful investigations upon the immunisation of cattle against tuberculosis at the Royal Veterinary College at Dresden.[2] Klimmer protests against sending out vaccine material in the dry, powdered state, on account of the great danger to the operator who attempts, in the field, to prepare this material for use. Klimmer's practice is to prepare suspensions of tubercle bacilli in the laboratory, just as has been done for a number of years at the laboratory of the Pennsylvania State Live Stock Sanitary Board.

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[1] "Bertrāge zur Experimentellen Therapie," Heft 9, 1905.

[2] "Berliner Tierärztliche Wochenschrift," 5th July, 1905.

The vaccine material that has been prepared in Dresden appears to be harmless to the vaccinated animals, and indications thus far are that it confers a serviceable degree of immunity, but it is not yet possible to draw final conclusions from this work for the reason that the vaccinated animals are still living, and it is impossible to determine positively whether they are free from tuberculosis. Klimmer proposes the use of a vaccine material made of mammalian tubercle bacilli that have been rendered less virulent by passage through a cold-blooded animal. The advantage from such a culture would lie in the total absence of danger to the operator.

Work upon the vaccination of cattle against tuberculosis at the laboratory of the State Live Stock Sanitary Board of Pennsylvania commenced in the year 1900. It has, therefore, been underway for about five years. We were able to show three years ago[1] that it was possible to increase the resistance of animals to tuberculosis to a very high degree by treating them with several intravenous inoculations of non-virulent tubercle bacilli of human type. Two cattle thus treated were inoculated intratracheally with a suspension of bovine tubercle bacilli, and both of them wholly resisted the inoculation. The only lesions in either of these animals was a slight thickening upon the wall of the trachea at the place where the hypodermic needle was inserted. Two unvaccinated animals, inoculated at the same time, in the same way, with the same quantity of bovine tubercle bacilli, from the same culture, became extensively infected with tuberculosis, showing the lesions on the mucous membrane of the trachea and bronchi, in the lungs, and in the bronchial, mediastinal, and postpharyngeal lymphatic glands.

The immunity that was obtained in the case of these two animals resulted from the administration of seven intravenous injections of vaccine material during a period of ten weeks. The doses of tubercle bacilli ranged from 13 to 26 mg. While this experiment, and other similar experiments, were sufficient to lead us to the conclusion that animals may be rendered immune to tuberculosis, it was at the same time evident that a process depending upon the application of vaccine material seven different times, would be of comparatively small value in general practice. Therefore, we set ourselves to the study of the effects of other methods of vaccination and to the development of a simple and effective method. During the past three years we have attempted to determine the immunising effects of several strains of tubercle bacilli. We have administered vaccines in different doses, at long and short intervals, and with varying number of doses.

For these experiments we have been supplied by the State Live Stock Sanitary Board with a large number of animals. For two years we have had the use of a farm where about one hundred cattle have been kept for experimental purposes. We have also had the use of stables at the veterinary department of the university of Pennsylvania, where from twenty to twenty-five cattle have been kept, together with numerous goats and small laboratory animals. Through the use of the farm we have been enabled to keep a considerable number of cattle under practical farm conditions.

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[1] "Philadelphia Medical Journal," 29th November 1902.

During the time that they have been under observation in our experiments, the resistance to tuberculosis of vaccinated animals has been tested through exposure to natural infection and by inoculation. When vaccinated animals have been exposed to infection and when they have been inoculated, unvaccinated, control animals have been equally exposed or inoculated. We regard this use of controls of the highest importance, because it is only in this way that a definite standard for comparison can be provided in order to measure the extent of resistance to a given amount of exposure. Moreover, it is only by the use of controls that one can ascertain whether exposure under natural conditions has been sufficient to lead to the infection of unvaccinated animals.

It is not enough to associate vaccinated cattle with tubercular cattle and then to conclude that the vaccinated cattle were immune because, when they were killed, they did not show lesions of tuberculosis. It might readily happen that the exposure was not sufficient to lead to the infection of unvaccinated cattle; but if, upon equal exposure, we find that the unvaccinated cattle are tuberculous, and the vaccinated cattle are free from tuberculosis, then we may safely and justly conclude that the vaccinated cattle received immunity from their vaccination. Our proof is based upon such clear evidence as this, frequently repeated.

Our experiments have shown that different strains of tubercle bacilli have different immunising values. Avian tubercle bacilli may produce an intoxication resulting in great emaciation, but avian cultures do not appear to confer upon animals so much immunity as results from the use of mammalian cultures that are non virulent for the animals upon which they are used. In order to produce artificial immunity in mammals it appears to be necessary to vaccinate with an attenuated mammalian culture.

We have not found any advantage either in respect to the degree of immunity or economy of time in the use of several vaccines made from cultures of different and progressive degrees of virulence.

The amount of immunity bears a rather definite proportion to the number of vaccinations and the amount of vaccine material used, provided however, that the animal be not "over vaccinated;" that is, that the vaccinations are not too close to one another, and that an excessive amount of vaccine is not administered. By over vaccination the resistance of an animal to tuberculosis may be reduced to a point below normal, or a fatal toxæmia may be caused.

Experiments have been made wherein animals vaccinated with different doses and different numbers of doses have been inoculated at the same time, and in the same way. While the resistance of all of the vaccinated animals to the inoculation has been much greater than the resistance of unvaccinated animals, it has been possible to observe a marked difference between animals vaccinated in different ways. For example, an animal vaccinated five times has more resistance than an animal vaccinated four times, and other conditions being equal an animal vaccinated four times has more resistance than an animal vaccinated three times.

From the practical standpoint, it is more important to ascertain the amount of vaccination that is necessary to protect animals under customary conditions of exposure and the shortest possible time during which a sufficient degree of immunity may be conferred, than it is to determine how great a degree of immunity may be produced.

Since it has been necessary to try a great number of different methods of vaccination, and since the time required for an experiment of this sort is much greater than is usual in experiments conducted through laboratories of hygiene, it has been difficult and time consuming to accumulate evidence upon which to base a general method for the vaccination of cattle.

It is already clearly evident that different degrees of immunity, and serviceable degrees of immunity, may be obtained at will. Under natural conditions, animals differ in respect to their inherited or acquired resistance to tuberculosis. Conditions of exposure differ, being very much greater upon some farms and in some herds than in others. Therefore, it seems to be reasonable to conclude that animals will require more artificial immunity to tuberculosis under some conditions than under others. The case is similar to the protection of cattle against anthrax by vaccination. In Pennsylvania we have found by experience, extending over a series of years, that the immunity conferred by vaccinating with the first and second vaccine of Pasteur usually is sufficient. On a few farms, however, the exposure appears to be more intense, and it is found that on such farms it is necessary to use the third vaccine of Pasteur. Experience may show that it will be necessary to vaccinate different breeds of animals, and animals kept under different conditions, in slightly different ways.

The amount of immunity that is conferred by three vaccinations is rather high, and will probably be sufficient in most cases. Indeed, two vaccinations may supply a sufficient amount of immunity under some conditions. Under other conditions it may be necessary to vaccinate four times.

The subject of the duration of immunity is very important, and is one that can be solved only by observations upon a large series of vaccinated cattle, some of which may be killed and examined *post-mortem* from time to time during a term of years. Of course it is necessary that animals kept for this purpose shall constantly be exposed to infection and unvaccinated controls must be similarly exposed, and the *post-mortem* results must show that the amount of exposure was sufficient to cause the infection of the unvaccinated animals.

Our own experiments indicate that artificial immunity may endure at least two years, and there is every reason to expect that additional experiments will show that immunity will be of longer duration than this. Perhaps it may last throughout the entire life of the animal; but conclusions on this point are entirely premature.

The effect of tuberculosis vaccination upon cattle already infected with tuberculosis has also received our attention[1]; it has been found that intravenous injections of tubercle bacilli of human type, non virulent for cattle. have a very marked influence not only in restraining the progress of existing lesions, but also in causing them to become encapsulated and to recede. In other words, such treatment appears to have a marked curative effect on some tuberculous animals. But this statement should be accompanied by a note of warning. We do not for a moment wish to imply that it is practicable, or even possible, at this time, to cure tuberculous cattle. The observations to be made in this direction are regarded by us as of importance chiefly as tending to throw additional light on the subject of immunity. It was shown that the treatment given had the same immunising effect upon infected animals as upon healthy animals. The effect of the immunisation in these cases was, first, to prevent the further dissemination of infection and, second, to cause the encapsulation and delimitation of the existing lesions.

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[1] "University of Pennsylvania Medical Bulletin," April, 1905. "American Veterinar Review," 1905.

It is probable that this method of treatment may prove to be of value with relation to young animals, or animals known to be but recently infected with tuberculosis. Such treatment will always have to be carried out with the most extreme care and under conditions that can be very fully controlled. It is not to be recommended for general practice.

As to the application of vaccination against tuberculosis; vaccination, properly applied, is effective, and we believe that it will prove to be of very great practical value. It is not possible on most American farms to establish a separate herd of infected cattle, and to keep the infected cattle as separate and distinct from the uninfected cattle as is necessary, if tuberculosis is to be treated according to the Bang system. The alternative, if tuberculosis is to be eradicated, is to destroy all of the animals that react to the tuberculin test. The latter plan involves a great deal of waste and loss, which, heretofore, has been unavoidable, and which has been amply justified because it has been less than that resulting from the continuation of the infection in the herd.

But it is important that a better, a less expensive, and, if possible, a more effective method shall be devised. It appears to be highly probable that vaccination may supply this method. It is admittedly impossible to test with tuberculin all of the tuberculous herds of a state. Therefore, it is not possible to discover and to either destroy or sequester all of the cattle that may be distributers of tuberculosis. Herds freed from tuberculosis by the use of the tuberculin test are always exposed to reinfection, and must be guarded by the double testing of all recruits and by systematic reinspections of the herd. All of this may be, and is, carried through successfully, and should be practised to a much greater extent than it is, unless an equally effective and cheaper method can be devised.

It will be a great step in advance to be able not only to discover that an animal is free from tuberculosis, but also to render that animal permanently immune to infection. It will be a great advantage to be able so to treat young cattle that they may resist tuberculosis even though they are continually exposed. The advantage to the breeder of being able not only to grow a herd free from tuberculosis, but immune to tuberculosis, will be very great. We believe that all of this may be looked forward to; but, until more experiments and observations are completed, vaccination should be applied only under such conditions as will permit exact observations to be made, and each vaccination must, for the present, be regarded as a scientific experiment.

THE QUESTION OF THE CO-RELATION OF BILIARY FEVER IN THE HORSE AND THE SUBACUTE FORM OF HORSE-SICKNESS.

By E. CLIVE WEBB, Lieut. A.V.D., Secunderabad, India.

DR EDINGTON, in his article on Biliary Fever in the Horse, Vol. XVIII., Part 1., *Journal of Comparative Pathology and Therapeutics*, endeavours to show that biliary fever of the horse and the condition to which he gives the term "malarial form of horse-sickness" are one and the same disease. This is the final conclusion to which his article leads as the result of evidence brought forward in the same, but I do not think that to many who have met with and had the opportunity of studying these diseases in their natural form the evidence brought forward will appear convincing.

In the report of Col. J. Nunn, A.V.D., of October 1888, referred to in the article, there are described four kinds of horse-sickness: (1) acute or pulmonary; (2) blue tongue; (3) dikkop; (4) subacute, or "what Mr Rutherford calls the bilious form." Continuing in the words of the report, "The subacute or bilious form comes on gradually, the animal being off his feed. . . . The bowels are constipated, and fæces coated with mucus. The urine is often of a deep amber colour, the temperature of the body from 105° to 108° F., and the pulse quick and hard." Allusion is also made to abdominal pains being frequently present and sometimes violent, and to fœtid diarrhœa and jaundice, and it is added that cerebral symptoms may be present, in which event the case is hopeless. Attention is directed to the fact that œdematous swellings appear on the body and limbs which contain a quantity of yellow serum, such swellings being regarded as of evil omen, the cases usually terminating fatally.

The description of these symptoms of the subacute form in Col. Nunn's report corresponds in such a marked degree with that of the form I described in an article on Horse-Sickness in the *Journal of Comparative Pathology*, Vol. XVI., Part 2, as the subacute intestinal or gastro-enteric, as to lead me to assume that my cases were of a similar nature to those described by Col. Nunn. To quote from my article: "Frequently in a careless examination the only symptoms that would be observable would be dulness and a tendency to lie down. This tendency to lie down is always present, and indicates a condition of uneasiness akin to colic. If a closer examination of the patient be made, the conjunctival membranes will be found to be very dark and intensely injected, showing petechial spots. The pulse will be found to be quickened and very weak. . . . The temperature is always raised, unless death be imminent. Death usually does not take place so rapidly as in pneumonic forms. . . . It is not always easy to diagnose cases of this class in their early stages, but the condition of the membrane is a good guide. In this country cases of fever with discoloured membranes are common, being probably malarial fevers, but it is possible to draw the conclusion that more animals than is at present supposed become affected with horse-sickness but recover in the early stages of the disease. Other symptoms more frequently shown in this class of case than in the pneumonic form are œdema of the tissues of the neck and filling of the supraorbital cavities. . . ." There is nothing pathognomonic in these symptoms, and they might equally well apply to a case of biliary fever.

Similarly, in the description by Mr Hutcheon of biliary fever, to which reference is made by Dr Edington, the symptons described would equally well apply to a case of the subacute form of horse-sickness, and the *post-mortem* appearance might easily be supposed to represent those of a case of the same.

As further evidence of the difficulty of diagnosis of these two diseases under discussion, I here quote from an article by Dr Theiler on Equine Malaria and its Sequelæ in the *Journal of Comparative Pathology and Therapeutics*, Vol. XVI., Part 2. Dr Theiler here gives a list of cases which succumbed to a secondary infection by a specific bacillus predisposed to by the piroplasma equi:—

"*Case I.*— . . . The animal died during the night.

A *post-mortem* was made early in the morning. The lungs were in a state of inspiration. The interlobular tissue was œdematous at the apices and roots of the lungs and at the base of the heart. The bronchi were filled with white foam. The blood was coagulated. In the right ventricle a solid clot was found, and there was a smaller one in the left one. A few ecchymoses were found in the endocardium. The spleen was much enlarged. The pulp, however, was fairly solid. The stomach was normal. In the small intestines a few red patches were seen, and injection of the blood vessels. The liver was jaundiced. A general slight jaundiced condition of all the organs was present. . . . From the state of the lungs some doubts were entertained whether this was not a case of "dunkop" (horse-sickness), although the season in which it occurred was against it. Some blood was therefore injected into a horse. Horse-sickness did not follow, however; and as the experimental horse died from a subsequent inoculation from horse-sickness it was proved that the lesions in the lungs of the former horse had nothing to do with this disease."

Several other cases of equine malaria with a somewhat similar confusing resemblance to horse-sickness are described in the same article.

Although by these several references to the articles of Mr Hutcheon, Col. Nunn, and Dr Theiler, I have shown the great similarity between the symptoms of this subacute form of horse-sickness and biliary fever or equine malaria, and the great possibility, clinically, of their confusion, yet the great similarity of these general pathological changes of two diseases, both presumably caused by parasites whose seat of growth and multiplication is in the general circulation, is no proof of the identity of the two diseases, any more than the identity of different septicæmias caused by bacilli specifically different although giving rise to the same general symptoms.

On reaching the final conclusion in the article it is sufficiently startling to tempt one to re-read, paying careful attention to the evidence brought forward in its support. Dr Edington was able to produce the disease by inoculation in animals born and bred locally, although he found coast animals more or less resistant. Dr Theiler, on the other hand, was unable to reproduce the disease by inoculation in country-bred animals running in the veldt, which failure he suggests may have been due to the fact that they have a high degree of immunity from having been tick infected and acquired immunity when young, as in the similar case of cattle born in redwater districts. Dr Edington also puts down the resistance of coast-bred animals to the fact that they are already infected, but since he claims that biliary fever is identical with the form of horse sickness designated by other observers "subacute or bilious," are we to believe that these coast bred animals continually harbour the demonstrable parasites of equine malaria, or the undiscovered virus of horse-sickness? From his conclusions one is forced to believe either that there is no such thing as subacute horse-sickness, and that the condition described as such by other observers was really biliary fever, or else that the piroplasma equi is the cause of horse-sickness, since one must assume that the same virus is the cause of all the forms of this latter disease.

The question naturally arises—does this disease designated by Dr Edington "the malarial form of horse-sickness or biliary fever" occur at particular seasons? One is given to understand, however, that it is continually present. This in itself is difficult to reconcile with all previous recorded history with regard to horse-sickness. Again, if this disease biliary fever is identical with a subacute form of horse-sickness, one would expect occasional transitional forms in which the subacute form approaches nearer in appearance to the acute form, and in some cases ends in acute symptoms, so that one would have expected occasional acute cases to keep cropping up during the time that the subacute form remained in existence, presumably the whole year round. With regard to his experiments, Dr Edington states that his first experience with this disease arose when he inoculated locally bred animals with the blood of a donkey inoculated with horse sickness blood ten days previously. He also readily produced it by inoculating clean horses with the blood of salted horses which had been inoculated with horse-sickness blood ten days previously.

A reasonable conclusion to draw from these experiments is that the donkey and horse from which the blood was taken to inoculate respectively the country bred animals and the clean horses were the harbourers of the piroplasma equi, with which they had become infected either naturally or by the horse-sickness blood used for their inoculation. There is no evidence to prove that these country-bred horses and clean horses had a mild attack of horse-sickness as the result of their inoculations. We are not told whether in the blood used for the inoculations any piroplasms could be detected, nor whether they were present in the inoculated animals during the febrile periods. In the next experiment, when two clean horses were inoculated with the blood of an unsalted horse which had been running on the veldt at night, parasites were demonstrated in the blood during the febrile period induced by the inoculation. Then follows an experiment as a corollary to the foregoing, viz.:—A horse was inoculated with blood whose virulence had been increased by the method of passage, and died as the result. The *post-mortem* appearances bore a striking resemblance to a case of horse-sickness, but I have already mentioned that this similarity of general symptoms is no proof of identity.

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Again, following this experiment is a reference to the fact that when a dose of serum taken from a horse which had recovered from horse-sickness, and been subsequently reinoculated on several occasions, was given to a horse in which the fever of horse-sickness was just beginning to appear or had already set in, a hæmaturia appeared about forty-eight hours later. After such a condition had appeared the animal died in less than forty-eight hours. The *post-mortem* examination in these cases presented appearances identical with those of the preceding experimental case of biliary fever.

Later follows the statement that the condition produced experimentally by Dr Edington was considered by him to be a malarial form of horse-sickness, agreeing with Col. Nunn's description of the "bilious form of horse-sickness."

So far as I have been able to gather, the arguments so far adduced to warrant the conclusion that the two diseases are identical are as follows:

(1) The admitted similarity between the *post-mortem* appearances of these two diseases.

(2) Practically the same as No. 1, viz., that in one experiment the horse dies as the result of inoculation with blood from a horse which has been running on the veldt, and which, although immune itself, harbours the piroplasm, rendered virulent by the method of passage; and, on the other hand, a horse which has received a dose of serum taken from a horse recovered from horse-sickness, and subsequently reinoculated at the time when symptoms of horse-sickness are commencing, dies and shows similar *post-mortem* appearances.

(3) In cases produced by inoculating with the blood of healthy horses, when they recovered and were inoculated with the blood of a recently reinoculated salted horse no fever resulted.

Against this scanty and inconclusive evidence relating to the identity of the two diseases we have the following facts to contend with.

(1) The occurrence of horse-sickness during a particular period of the year only, and its usually abrupt termination after the first frost, whereas cases of biliary fever may occur at any season.

I am inclined to think that the cases described by Col. Nunn, since they were described in the report on horse-sickness, are more probably identical with the cases described by myself under the heading of subacute or gastro-enteric form. And Col. Nunn's cases probably only occurred during the horse-sickness season, otherwise no doubt special notice would have been taken of them; and although it is possible that a coincidental case or two of actual biliary fever may have occurred during the particular season in which the cases I described occurred, and may have been mistaken for subacute horse-sickness, the fact that when the "season finished" no more cases of this subacute form occurred precludes the suggestion that the majority of them may have been biliary fever.

(2) I consider that a very important *post-mortem* appearance in subacute as well as acute cases of horse-sickness, and a lesion which is important in distinguishing *post-mortem* between biliary fever and subacute horse-sickness, is the intense inflammation of the mucous membrane of the left half of the stomach which one gets in the latter disease (first noted by Prof. M'Fadyean).

(3) Biliary fever not only occurs at any season during the year but has also been reported as occurring in other countries, *e.g.*, India, where the piroplasma equi has been demonstrated in the blood. In a report on dourine, by Dr Lingard, in which he deals also with piroplasmosis, he states that he found the piroplasma equi in several apparently healthy horses, which were brought from certain districts for experimental purposes. As there is no horse-sickness in India, I do not see how one can reconcile such a conclusion as to its identity with biliary fever.

(4) The presence of the piroplasma equi and its demonstration in the blood will confirm a diagnosis of biliary fever, although it may be difficult or impossible to find it at every examination made, but according to Dr Theiler it is most easily found during the early stages of the disease and during the height of fever. It is not impossible, however, that the two diseases might be running concurrently, in which case a confirmation of the diagnosis of horse-sickness would only be possible after death by inoculation of a clean horse with the blood of the patient.

Finally, I submit that according to our present knowledge subacute horse-sickness and biliary fever must be regarded as separate and distinct diseases, each caused by its specific organism, the former as yet not demonstrated, and the latter the piroplasma equi.

SURRA TRYPANOSOMA IN CATTLE.

By MAJOR H. T. PEASE, I.C.V.D.

IF I may be permitted, I should like to say that Captain Martin is a little behind the times *re* priority of discovery of the surra trypanosoma in cattle. So long ago as 1892 Dr Lingard inoculated cattle with surra from the horse, and published detailed information regarding the course of the disease in these animals. Again, in 1896, Lingard, reporting on an outbreak of Surra at Kurnal, details of which were published in the *Veterinary Journal*, remarks, when dealing with the loss of bullocks which occurred in the depot, as follows: "On the 14th December, in company with Lieutenant Eassie, I visited the bullock hospital and picked out two bullocks, Nos. 13 and 152, as being likely subjects of surra. On the morning of the 15th specimens of blood from both these animals were submitted to microscopical examination, and the surra hæmatozoon was found to be present in both. Bullock No. 13 died on the 16th December." Since this time I suppose that everyone who has looked has found the trypanosoma in cattle from surra tracts. I personally have found that a buffalo inoculated with a trace of surra blood from a camel, after passing through two or three paroxysms, apparently recovered, but the blood still contained the trypanosoma in small numbers, and was capable of reproducing the disease in dogs fifteen months after apparent recovery. There was no sign whatever of illness, and the buffalo remained in perfect condition. Lingard in 1891 showed that the blood of bullocks apparently recovered was capable of producing the disease some months afterwards. I feel sure that Major Raymond would be the first to disclaim any credit for a discovery which did not belong to him. The matter is really of very little importance, but, as it has been prominently brought forward in this *Journal*, it is perhaps well that the sequence of events should be correctly stated, so far as India is concerned. The statements made can be verified by a reference to Lingard's *Surra Report*, Vol. I., and the *Veterinary Journal* for 1896 or 1897.

A REPLY TO CAPT. MARTIN ON TRYPANOSOMIASIS.

By Capt. J. D. E. HOLMES, M.A., M.R.C.V.S., I.C.V.D.

THE corrections which Capt. Martin kindly offered in the note which appeared in the last number of the *Journal of Comparative Pathology and Therapeutics* seem to me rather unnecessary and most misleading. Capt. Martin objects to the statement that "up to the present it has not been demonstrated that the cattle of India suffer from trypanosomiasis," and informs us that Major Raymond had shown him trypanosomes in the blood of cattle as far back as 1898, etc. I do not doubt Capt. Martin's word, but I am not aware of any publication on this point. Has any accurate description of the trypanosome been given to identify it as the trypanosoma evansi? Can Capt. Martin offer any evidence in support of his statement that "the spontaneous occurrence of trypanosoma evansi in cattle in India has been well known for years" Is this alleged fact confirmed by any reliable authority, or is it merely a "matter of general knowledge"? As far as I know there are absolutely no grounds for such an assertion, nor have I any reason to believe that such an opinion is prevalent in India.

There are also grounds for condemning an opinion of this kind as erroneous. Inoculation experiments on cattle with the trypanosoma evansi carried out by Lingard show that the parasites apparently do not persist for any length of time in the blood stream, and after some weeks they disappear and cannot again be demonstrated. The blood of these experimental cattle was examined daily for over two years with negative results.

Capt. Martin attempts to throw ridicule on the suggestion that the outbreak among the cattle of Mauritius was not caused by the trypanosoma evansi. Without any consideration of the question, he condemns us for our audacity in venturing an opinion contrary to that expressed by Laveran and Nocard. To any one who has followed the most recent work on this subject there is nothing startling in the suggestion. It is, in fact, in strict accordance with the findings of an investigator second to none in the field of trypanosomiases.

Novy has shown that the apparent similarity of morphology is not sufficient for the correct classification of trypanosomes, and that the cultural test is the more reliable one. As our knowledge stands, there are no satisfactory grounds for connecting the trypanosoma evansi with any other disease than the one known as surra of horses.

Capt. Martin is not satisfied with accusing us of robbing Major Raymond of his discovery of trypanosomiasis in cattle, but also finds us guilty of indirectly claiming priority for Stockman in the finding of piroplasms in the blood of cattle of India. On page 319 I mention the work done by Major Raymond in 1898 before speaking of Stockman's investigations in 1902.

As regards the contradiction which Capt. Martin points out on page 209, I should have thought that what I wished to express would have been clear to the average intellect. The word "affected" is here used in a broader sense than to convey the meaning of "infected" only. In the second sentence I state the temporary infection and speak of the general health as not being affected.

SUGGESTIONS FOR A UNIFORM SYSTEM OF INTERPRETING THE TUBERCULIN REACTION IN CATTLE.[1]

By Prof. Dr A. EBER, Leipzig.

BY the tuberculin "reaction" is generally understood a rise in bodily temperature amounting to fever, which rise is accompanied by more or less acceleration of the pulse and breathing, less frequently by rigors, loss of appetite, and other visible signs of disturbance in the bodily functions. In adult cattle these secondary symptoms are less marked and are sometimes entirely absent. A "local reaction" (swelling) is not invariably to be expected, for experience shows that when it is possible to avoid irritating the skin at the point of inoculation (by clipping and shaving away the hair, washing with sublimate or strong alcohol) it fails to occur. As a consequence, the only reliable criterion of reaction is to be found in a distinct rise in bodily temperature amounting to fever.

What is to be Regarded as a Febrile Reaction in Cattle?

At the present moment we are not in possession of generally-recognised uniform data on which to base our answer to this question.

A priori we may assume one of two logical positions :—

(1) The decision may be based on the highest observed temperature after reaction, without reference to the animal's actual temperature before injection or to any arbitrarily fixed temperature.

In this case the highest temperature to be regarded as normal is fixed upon to begin with, and any rise beyond is regarded as a reaction.

(2) The decision may be based on the rise in temperature relatively to that observed before injection or to an arbitrarily fixed temperature.

In this case a certain rise in temperature is fixed upon as the utmost which can be regarded as falling within the normal limits of variation, and anything exceeding this, without reference to the absolute temperature attained, is regarded as a reaction. In this case different conclusions are possible, depending on whether we adopt as our basis the highest temperature recorded before injection, the average temperature of a series of readings, or the particular temperature existing at the moment of injection.

As a general rule, it is customary in medicine when using the first method to solve the question by comparing the highest observed temperature with a scale of normal temperatures compiled on the basis of prolonged observation for each species of animal. Having regard, however, to the wide limits within which the ox's temperature may normally vary, it has always been a matter of great difficulty to fix a precise scale. The growing use of tuberculin in the early nineties rendered this question a very important one, and owing to its difficulty observers were more and more driven to base their conclusions on the relative rise in temperature, *i.e.*, to adopt method No. 2.

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[1] From the "Proceedings of the VIIIth International Veterinary Congress," Budapest.

As a general rule the rise was measured as against the highest temperature recorded in a series of observations made before injection. The difference between the highest temperature before injection and the highest after was compared with a fixed standard of permissible variation, and the conclusion was arrived at in a purely mechanical way, *i.e.*, as to whether or not a given rise should or should not be held to constitute a reaction.

Most of the tests carried out for magisterial authorities during the early nineties were based on this method. The limits of "permissible variation" in bodily temperature were not then authoritatively fixed.

This method, however, is only free from objection when either a marked rise of temperature occurs or the rise is clearly of no importance. On the other hand, when the rise is of a moderate character only, the decision must largely be influenced by chance factors. And the fact that several measurements of temperature are necessary in order to fix a reliable medium temperature before injection is not precisely calculated to simplify the question.

The commencing struggle against tuberculosis about the middle of the last decade rendered it therefore increasingly necessary to discover the cause of the varying results given by tuberculin, and to provide fresh principles on which to interpret the temperature curves resulting from tuberculin injection.

The investigation was greatly facilitated by the fact that meanwhile the numerous tests made in large establishments had resulted in the collection of a great body of statistics, from which "normal limits" for the bodily temperature of cattle could be deduced, and the correct conclusions drawn from the highest observed reaction temperatures without reference to the pre-injection temperature.

It is therefore scarcely surprising that since the middle of the nineties most authorities have discarded the "relative rise" method, in favour of the "absolute rise," utilising for the purpose scales of temperature based on numerous and prolonged observations on cattle, and only falling back on the "relative rise" method in isolated special cases.

This method also seems to me best adapted at the present time as a basis for formulating a uniform system of interpreting the tuberculin reaction in cattle. Not the least recommendation is the fact that in this method the operator has need only to take the temperature *once* before injection, in order to ascertain if the animal is free from fever.

Reviewing the numerous publications on this head made since the middle nineties, it is satisfactory to find that most writers are unanimous in regard to many points in the interpretation of the temperature tables when using this method.

The following conclusions are strongly supported:—

(1) Any cattle, which at the moment of injection are free from fever (*i.e.*, whose temperature does not exceed 39·5° C.), and which

subsequently exhibit a rise above 40° C., are to be regarded as having "reacted."

(2) In cattle up to six months of age, a rise not exceeding 40° C., and in cattle of greater age a rise not exceeding 39·5° C., is to be regarded as not warranting suspicion.

It would appear that essential differences of opinion would only occur in the case of rises of temperature between 39·5° C. and 40° C. in cattle older than six months.

Of such cases, however, most authors agree in regarding as reactions those in which the total rise above the pre-reaction temperature amounts to or exceeds 1° C.

The only doubtful cases, therefore, are animals over six months old in which the temperature rises to a point between 39·5° C. and 40° C., and in which the difference between the highest temperature attained and the temperature before reaction is less than 1° C.

To dispose of these cases, however, without further test as "not warranting suspicion," as is recommended in the new Prussian regulations regarding the use of tuberculin, seems to be a mistaken policy, and I hold to the opinion that it is scientifically preferable to group them together as suspicions.

The number of these reactions will undoubtedly be affected by the manner in which the tuberculin test is carried out. My experience suggests that doubtful reactions will be rarer the sooner temperature measurements follow on the injection, the more frequent they are, and the longer they are persevered in. Nevertheless, they cannot be entirely avoided, especially when dealing with large groups of animals.

Experience shows that the "doubtful" group includes both tuberculous and non-tuberculous animals, and that the means at present at our disposal are insufficient to admit of precise distinction between them.

If the severity of the reaction were in direct proportion to the extent of the tuberculous changes, it would be easier to contemplate the results of any errors arising from faulty diagnosis (such, for example, as the strict application of the above-mentioned Prussian order would have), for at the worst only a slightly tuberculous animal would be passed as healthy. But experience shows that the animal which reacts but slightly is often found on *post-mortem* examination to have extensive tuberculous lesions. It is very clear, then, that to entirely pass over or to wrongly interpret the reactions lying between 39·5° C. and 40° C. may be fraught with serious consequences.

In the numerous tuberculin tests I have carried out, I have always regarded those animals of the so-called "doubtful reaction" group whose highest temperature reading exceeded the primary temperature by at least ·5° C., and whose temperature curve showed the unmistakable characteristics of fever (gradual rise, pause at the summit, and gradual decline), as having reacted. Those animals of the doubtful group, however, whose highest temperature reading fell short of a total rise of ·5° C., or whose reading, whilst exceeding ·5° C., showed only isolated, uncoordinated rises, I regarded as not reacting. At the same time—and I lay special emphasis on this—the condition of the animals was also tested by a thorough clinical examination.

Whoever practices this method of diagnosis, utilising every means of control that offers, and checking his results by *post-mortem* examination, will soon attain such skill in judging of the so-called doubtful reactions as to be able to distinguish with a very fair degree of certainty between the tuberculous and the healthy.

In those countries in which an attempt has been made to combat tuberculosis by the use of tuberculin it is easy to adopt the method, practised in Denmark, of leaving the final decision of doubtful cases to the same central organisation as is entrusted with the business of stamping out the disease. This method has the further advantage that the experience acquired can thus be best utilised for the general good.

I am therefore of opinion that the so-called doubtful reactions are not beyond the scope of scientific exact investigations, and that the decision as to which animals are suspicious and which non-suspicious is not to be settled by schematic formulæ, but by the application of the experience acquired by study of individual cases.

Nevertheless, circumstances exist under which it is desirable to have available a simple formula which will allow us promptly, without difficulty and with a fair degree of certainty, to descriminate between suspicious and non-suspicious cases. Ostertag's proposed rule conforms to these requirements, viz., that any animal over six months of age whose temperature rises above 39·5° C. and exceeds by ·5° C. the highest temperature shown before injection should be regarded as having reacted.

Kühnaus goes a step further, and views any rise of temperature above 39·5° C. in adult cattle as a reaction.

Both propositions have in practice the great advantage of providing a simple easily-understood method of distinguishing between reacting (suspicious) and non-reacting animals whilst avoiding the bugbear of doubtful reactions. Nevertheless there is no doubt that this facility of diagnosis is only attained at the cost of reliability, and that every now and then an entirely healthy animal will be condemned as tuberculous. The possibility of such an occurrence must naturally be somewhat greater when using Kühnaus' than when using Ostertag's formula, for which reason it would be well to insist on a minimum rise of ·5° C. However this may be, mistakes resulting from the use of these simplified rules of diagnosis are less serious as regards the stamping out of tuberculosis than errors in the opposite direction, leading to the introduction of tuberculous animals into healthy herds. It depends very largely, therefore, on what object one has in view in applying the tuberculin test whether one should employ the simplified formulæ of Ostertag and Kühnaus, or institute an investigation on the basis of the above described principles. In my opinion, however, an investigation of the above kind can scarcely be omitted when the tuberculin test is invoked to determine whether or not a given animal should be slaughtered.

As cattle less than six months old seldom suffer from extensive tuberculous changes, it is necessary, in their case, to deal with doubtful reactions in a special way.

According to Ostertag, all rises in temperature above 40° C. in animals less than six months old may at once be set down as reactions, provided the highest temperature after injection exceeds the highest temperature before injection by at least ·5° C. As long as one observes the precaution not to submit to the tuberculin test any animal of this age, whose temperature exceeds 39·5°, there only remains the measurement of the relative rises in temperature. Nevertheless, the strict observance of this rule in young cattle, whose temperature is known by experience to be higher than that of older animals, may occasionally give rise to difficulties.

On the basis of the foregoing propositions I make the following suggestions for the formulation of uniform principles for interpreting tuberculin reactions in cattle.

(*a*) For young cattle up to the age of six months:—

In young cattle, up to the age of six months, whose temperature previous to tuberculin injection does not exceed 40° C., any rise above 40° C. is to be regarded as a reaction, provided the difference between the highest temperature before injection and the highest after amounts to at least ·5° C.

(*b*) For cattle above six months of age:—

(1) No animal whose temperature exceeds 39·5° C. to be tested.

(2) Rises of temperature to 39·5° C. after injection to be regarded as non-suspicious.

(3) Every animal whose initial temperature is below 39·5° C., and which shows a rise above 40° C., to be regarded as suspicious.

(4) Rises in temperature between 39·5° C. and 40° C. to be accounted reactions, provided the difference between the highest pre-injection and the highest post-injection temperature amounts to at least 1° C.

(5) Rises in temperature between 39·5° and 40° C. to be considered as doubtful in cases where the difference between the highest pre-injection and the highest post-injection temperature is less than 1° C. Such cases to be separately considered.

The separation of these cases into reacting and non-reacting to be otherwise determined. In this connection valuable indications are to be found in a comparison between the highest pre-injection and post-injection temperatures, which as a rule in reacting animals should amount to at least ·5° C.; in the character of the temperature curve, which in reacting animals should display the regular fever form; and in the results of a careful clinical examination, which should be instituted in all doubtful cases.

(6) In all cases met with in current practice, where the tuberculin test is only invoked in order to ascertain the extent of tuberculosis in a herd or to separate tuberculous from non-tuberculous animals for the purpose of fighting tuberculosis, it is sufficient to accept Ostertag's formula, and regard all cattle as suspicious in which an injection of tuberculin causes the temperature to rise above 39·5° C., and in which the highest post-injection temperature exceeds the highest pre-injection temperature by at least ·5° C.

FURTHER NOTES ON PIROPLASMOSIS OF THE HORSE, MULE, AND DONKEY.

By Dr A. THEILER, Bacteriologist to the Government of the Transvaal, Pretoria.

INOCULABILITY OF EQUINE PIROPLASMOSIS.

THE description of the piroplasma found in horses, mules, and donkeys was fully entered into in my previous communications on the same subject. Symptoms and *post-mortem* lesions observed in this disease in mules and donkeys, as encountered in the field, were dealt with in my annual report for the year 1903-04. At that time already I expressed the opinion that the piroplasmosis in the three mentioned equines was identical, due to the same species of piroplasma—viz., piroplasma equi (Laveran). The conclusive proof—viz., that of producing the disease in the horse with virulent mule or donkey blood or *vice versa*—was at that time wanting. I had only one observation at my disposal—that showing the inoculability of equine piroplasmosis from an immune horse into a susceptible one by means of defibrinated blood of the former one. This observation indicated the chronic piroplasmosis, where the blood of immune animals proves to be infective for susceptible animals of the same species. Having failed in former experiments to transmit the piroplasmosis of the donkey into the horse, because only country-bred or well acclimatised horses were obtainable, recourse was had this time to the importation of horses, mules, and donkeys from the Argentine Republic, which animals are, as we know from practical observations, very susceptible to piroplasmosis. They were kept stabled in the premises of the laboratory in order to exclude any possible natural infection. The infective material was derived in every instance from immune animals. The first experience with the blood of the immune horse having proved successful, the same could be expected from immune mules and donkeys. The object of the experiments was not solely directed to demonstrating the identity of the piroplasma in the various equines, but also to test the possibility of an inoculation similar to the one in redwater. The experiments also give us more useful information about the incubation, course of the disease, and its immunity, and will prove it so closely allied to redwater in cattle that more than a similarity must be expected, although an identity has to be excluded.

(*A*).—*Experiments to Prove that the Inoculation of Blood of a Horse Immune against Equine Piroplasmosis produces Piroplasmosis in Susceptible Horses.*

Argentine horse, about four years old, brought directly from board ship to the premises, was injected on 3rd October 1904, both intrajugularly and subcutaneously, with 10 cc. defibrinated blood of a horse, No. 523, which in July of the same year had recovered from a spontaneously acquired attack of biliary fever, during the fever reaction of which piroplasma equi was very frequently found in the blood. After an incubation period of six days—viz., the 9th October—the temperature rose, and on the seventh day piroplasmata were present in the blood. These parasites increased within twenty-four hours, after which time rosettes were especially numerous (11th October 1904). Red urine was voided for the first time on 11th October 1904, and it was noted during the following few days. A spectroscopical examination, both in my laboratory and in that of Dr Ingle, Chief of the Chemical Division, proved the exclusive presence of oxyhæmoglobin, which was still present in traces after the urine had cleared up and taken a more brownish colour. On 15th October 1905 a decrease in the number of parasites was distinct, and at the same time a slight poikylocytosis made its appearance. The decrease continued, and on 20th October the parasites had disappeared. The symptoms which were observed during the course of the disease were, besides red urine, a strongly pronounced jaundice, a rapid loss of condition, accelerated respiration, and rapid pulsation of the heart. The quickened breathing kept on for some time after the temperature had returned to a more or less normal level. The examination of the blood was discontinued. A slight secondary reaction was noticed, starting twenty-four days after inoculation and lasting about ten days. As no microscopical examination was made during that reaction, it is impossible to say to what it was due.

Argentine, No. 598, about four years old, injected on 4th November intrajugularly with a mixture of defibrinated blood of Horses 442, 523, 599, all of which had passed through an attack of biliary fever, No. 442 on 19th to 27th September 1904. A reaction began four days later, but did not reach a high elevation. The piroplasma equi could only be traced during the descending period of the fever, at which time rosettes were found. Then followed a period of six days, when the temperature was normal and during which no piroplasmata were seen, to be succeeded by a high elevation of the fever, the reappearance of piroplasma equi, and an irregular temperature curve which goes over after seven days into normal. In this horse the peculiarity was noticed that during the first fever reaction the round piroplasma was principally noticed; while during the second one quite an atypical form was seen, more oval, and even willow-leaf shaped, and twin parasites in one and the same red blood corpuscle, as in the case of piroplasma bigeminum.

Argentine horse, 603, about four years old, was injected on 28th November 1904 with defibrinated blood of Horses 442 and 599—15 cc. under the skin and 15 cc. into the vein. Piroplasma equi was noticed in this horse on 6th December 1904, when rosettes were already present and at a time when fever reaction had not yet properly started. The parasites were present during the next five days, when they disappeared completely. The animal had been treated with a subcutaneous injection of quinine, and at the place of injection, as is frequently the case in biliary fever, a local phlegmon formed, which is responsible for the continuance of the fever. The following tabulated record of the number of red corpuscles during the disease shows their decrease and increase:—

Date.	*Number of Red Corpuscles.*	*Presence or Absence of Piroplasma.*
29/11/04	7,200,000	
30/11/04	6,610,000	
1/12/04	6,260,000	
2/12/04	6,400,000	
3/12/04	6,080,000	
4/12/04	5,840,000	
5/12/04	5,880,000	
6/12/04	5,080,000	Piroplasma equi appears. Rosettes.
7/12/04	4,792,000	Piroplasma fairly frequent. Rosettes and round, oval, and flagellated forms.
8/12/04	3,420,000	Piroplasma rarer.
9/12/04	2,320,000	Piroplasma still rarer; some bacillary shaped forms are visible.
10/12/04	2,888,000	Examination negative.
11/12/04	2,764,000	Piroplasma equi very rarely met with.
12/12/04	3,552,000	Piroplasma absent.
13/12/04	4,566,000	" "
14/12/04	5,936,000	" "
15/12/04	6,160,000	" "
16/12/04	6,552,000	" "
17/12/04	6,496,000	" "

During this time the usual characteristic symptom of equine piroplasmosis, viz., general jaundice, was present, and a rapid loss of condition had taken place. The horse had apparently recovered, when, on 21st December, a second attack of fever occurred, which lasted five days and to which the animal succumbed. During this reaction round and willow-leaf shaped parasites were noticed and twin forms were also met with.

Post-mortem examination revealed a slight jaundiced condition of the serous membranes of the body. There were petechiæ on the endocardium of the left ventricle and also on the epicardium. The liver was enlarged but of normal colour. The spleen was but slightly enlarged. The remainder of the organs seemed to be normal.

Argentine horse, No. 597, about four years old, was injected on 28th November 1904 with 10 cc. blood of horse 523.

In this case piroplasms were seen before there was an elevation of temperature, which elevation took place late and lasted but four days. The piroplasmata were but rarely met with, but the symptoms of an anæmia were well pronounced by an intense pallor of all the visible mucous membranes. There was no jaundice present. The following tabulated record shows the variations in the number of the red corpuscles during the reaction :—

[TABLE.

Date.	*Number of Red Corpuscles.*	*Remarks.*
29/11/04	6,080,000	
30/11/04	5,144,000	
1/12/04		
2/12/04	3,536,000	
3/12/04	3,948,000	
4/12/04	3,728,000	
5/12/04	3,984,000	Piroplasma equi present
6/12/04	4,652,000	„ „ but rare
7/12/04	4,888,000	„ „ „
8/12/04	4,884,000	„ „ „
9/12/04	4,954,000	„ „ very rare
10/12/04	3,416,000	„ absent
11/12/04	2,496,000	„ „
12/12/04	3,052,000	
13/12/04	2,488,000	
14/12/04	3,408,000	
15/12/04	5,238,000	
16/12/04	5,392,000	
17/12/04	6,132,000	

On 30th December 1904 this horse was subjected to the simultaneous horse-sickness inoculation. It passed through an attack of dikkop and recovered, the temperature returning to normal, but on 16th January 1905 the temperature rose suddenly again, piroplasma equi reappeared in the blood, and the animal died on 23rd January.

The *post-mortem* showed a fair condition of the carcase. There was a yellow discoloration of the serous membranes, a subcutaneous collection of yellow liquid, and the lungs were œdematous and somewhat pale in colour. The epicardium was diffusely reddened by petechiæ, and large ecchymoses were found on the endocardium of both ventricles. The liver was much enlarged and yellow, and the central veins were filled with blood. The spleen was but slightly enlarged and rather firm. The kidneys were pale. The lymphatic glands of the various regions were enlarged.

Argentine horse, No. 606, about four years old. Injected on 28th November 1904 with 30 cc. of a mixture of blood of horses Nos. 442 and 599, 15 cc. subcutaneously and 15 cc. into jugular vein. After an incubation period of five days the temperature rose, and on the following day the symptoms of jaundice were already noticeable in the eye. Piroplasmata were very numerous during the reaction, which lasted ten days and ended with death on the 13th December 1904. The *post-mortem* revealed the typical picture of equine piroplasmosis with general jaundice.

Conclusions.

(1) Equine piroplasmosis is inoculable with blood of an immune horse into a susceptible horse, the Argentine horse proving very suitable for this purpose.

(2) The piroplasmosis produced by an inoculation with immune blood has an incubation period ranging from five to six days.

(3) In cases of recovery from the first attack of piroplasmosis a second attack is usually observed, during which the piroplasma equi is less frequently found than in the first attack, and is also changed in shape, usually exhibiting the willow-leaf form. Death may result from either the first attack or the second one, but is more frequent after the latter.

(4) The pathological change caused by the presence of piroplasma equi is an acute anæmia due to the destruction of red corpuscles.

(5) The intrajugular injection of blood from an immune horse into a susceptible one proves to be very dangerous, and offers but little prospect of a practical inoculation.

(*B*).—*Experiments to Show that the Inoculation of Blood of a Horse Immune against Equine Piroplasmosis produces the Disease in Susceptible Mules.*

Argentine mule, No. 587, about three years old, directly imported from board ship in Durban, was injected on 4th November 1904 with a mixture containing equal parts of defibrinated blood of horses 523, 599, and 442, viz., 15 cc., into the jugular vein and 15 cc. under the skin. On the fifth day a first rise of temperature took place, but the examination of the blood remained negative; it was followed up by a second reaction during which piroplasma equi was present. After the lapse of about ten days, during which time the examination of the blood gave negative results, another reaction took place, during which the piroplasma equi made again its appearance. After that no further disturbance was noticed in the temperature-curve. This animal showed during the first reaction an exquisite paleness of the mucous membranes and lost considerably in condition.

Argentine mule, No. 589, about three-and-a-half years old. Same origin as above. Injected on 28th November 1904 with a mixture of equal parts of blood of three immune horses, Nos. 442, 523, and 599. A first slight disturbance took place on 4th December 1904, viz., six days after injection. Piroplasma equi was seen the next day, it subsequently increased in numbers, and during the 8th and 9th December 1904 rosettes were fairly frequent. From the 10th December 1904, when the temperature had reached its highest point, the parasites became rarer, and at the same time some atypical bacillary-shaped ones appeared. The next day, viz., 11th December 1904, the parasites had almost vanished from the blood. After a lapse of about seven days a second reaction took place, starting on 19th December 1904, during which unfortunately no examination took place.

Mule 596. About three years old. Same origin as previous one. It was injected on 28th November 1904 intrajugularly with 10 cc. blood of horse 523. On the eighth day after inoculation a slight rise in temperature occurred, and the following day piroplasma equi appeared in the blood. The reaction was never high and the parasites were very rarely seen. Also in this case a second reaction occurred a few days after the first one had abated, the examination of the blood during which gave negative results. The following gives the record of the number of red corpuscles during the first reaction :—

Date.	*Numbers of Red Corpuscles.*	*Remarks.*
5/12/04	5,792,000	
6/12/04	6,448,000	
7/12/04	6,600,000	Piroplasma present, but rare
8/12/04	6,712,000	„ „
9/12/04	6,140,000	„ „
10/12/04	4,392,000	„ „
		Mucous membranes pale
11/12/04	5,312,000	Piroplasma present
12/12/04	4,948,000	
13/12/04	4,728,000	
14/12/04	6,500,000	
15/12/04	6,336,000	
16/12/04	6,720,000	
17/12/04	6,832,000	

Mule 592. About four years old. Same origin as previous one. Injected on 28th November 1904 with a mixture of blood of horses 599 and 442, viz., 15 cc. subcutaneously and 15 cc. intrajugularly. After a lapse of five days a slight rise of temperature was noticeable, during which the piroplasma equi was fairly frequent; then a distinct high reaction set in, lasting from 8th to 14th December 1904, and during which the piroplasma was but rarely noticed. Also in this case a second although not very characteristic reaction took place, but no parasites were seen. The following tabulated form gives a record of the number of red corpuscles during the first reaction:—

Date.	*Numbers of Red Corpuscles.*	*Remarks.*
29/11/04	6,580,000	
3/12/04	7,012,000	
5/12/04	5,816,000	
6/12/04	6,120,000	Piroplasma fairly frequent.
7/12/04	6,698,000	
8/12/04	5,400,000	
9/12/04	4,528,000	Piroplasma rare
10/12/04	3,064,000	„
11/12/04	3,928,000	„
12/12/04	4,376,000	
13/12/04	4,072,000	
14/12/04	5,048,000	
15/12/04	4,908,000	
16/12/04	5,000,000	

Mule 595. About four years old. Same origin as previous one. Injected 16th December with 10 cc. defibrinated blood of horse 597. A slight reaction took place, and on the ninth day piroplasma equi was noticed in rare numbers. In this instance no second reaction was noticeable.

Conclusions.

(1) Equine piroplasmosis is inoculable with blood of an immune horse into a susceptible mule, Argentine mules proving very suitable for this purpose.

(2) The incubation period of piroplasmosis in mules corresponds with that found in horses.

(3) All inoculated cases ended in recovery. As in horses, two reactions are observed, the second one being characterised by the scarcity or complete absence of parasites.

(4) The inoculated piroplasmosis is in mules not so fatal as in horses, and the anæmia is also less pronounced.

(5) The inoculation of blood from an immune horse into a susceptible mule can be made use of as a means of preventive inoculation against piroplasmosis.

(C).—*Experiments to Show that the Inoculation of Blood of a Horse Immune against Piroplasmosis produces this Disease in Susceptible Donkeys.*

Donkey 736, about four years old, imported from the Argentine and brought by rail from Capetown off the boat, was injected with blood of horse 597. There was a distinct primary, followed up shortly afterwards by a secondary, reaction. By some mistake the blood of this donkey was only examined after the secondary reaction had passed. Piroplasma equi was therefore not found in this case. The donkey lost rapid condition, went down, and was unable to rise again. He died on 12th February 1905.

The *post-mortem* revealed a slightly enlarged spleen with softened pulp. There was a slight increase of liquid in the pericardium and *ante-mortem* clots in both ventricles. These lesions speak for piroplasmosis, viz., the chronic form of this disease.

Donkey 739 and donkey 740. Both injected subcutaneously on 21st March 1905 with 10 cc. blood of horse 724. Both donkeys showed a typical fever reaction, during which the piroplasma equi was found, viz., in donkey 739 on 27th and 28th March 1905, and in donkey 740 on 29th and 30th March 1905. The reaction in both cases was very slight, and could only be diagnosed as piroplasmosis by microscopic examination of the blood.

Conclusion.

The injection of blood of a horse immune against piroplasmosis into a susceptible donkey produces piroplasmosis in the donkey.

(D).—*Experiments to Show that the Blood of a Mule Immune against Equine Piroplasmosis remains infective for Susceptible Horses.*

Horse 724, about four years old, imported from the Argentine, landed in Capetown, and brought from board ship directly by rail to Pretoria. It was inoculated intrajugularly on 16th January with 10 cc. defibrinated blood of mule 592. After three days a reaction began and lasted five days. Piroplasma equi was noticed for the first time on 22nd January, and disappeared after the temperature had returned to normal.

The animal showed all the symptoms of biliary fever, and had lost in condition, going down and being unable to stand for some days. From the 14th to the 23rd February a second reaction took place, during which, however, no piroplasmata were seen. This second reaction reduced the animal to a skeleton.

Horse 721. The same origin as above. It was injected subcutaneously on 15th February with 10 cc. blood of mule 592. A reaction began on the fifth day, but the temperature did not go high, its culminating point being 104° F. Piroplasma equi was present on 23rd February and during the following days up to the date of death, which occurred on 26th February.

The *post-mortem* was made soon after death. There was a slightly jaundiced condition of the serous membranes. The spleen was about double its normal size, with softened pulp. The liver was dark-brown and strongly congested. The kidneys showed the colour of sepia.

Conclusions.

(1) Equine piroplasmosis is inoculable from an immune mule into a susceptible horse.

(2) The piroplasmosis of horses produced by immune blood of mules is of a very grave character, and there is little prospect that immune mule-blood can be utilised for protecting susceptible horses.

(E).—Experiment to Show that the Blood of a Mule Immune against Equine Piroplasmosis remains infective for Susceptible Mules.

Mule No. 593. Argentine, about four years old. Brought directly from board ship to the laboratory, and injected on 16th January 1905 with 10 cc. defibrinated blood of mule 597 into the jugular vein. From the sixth day a disturbance of the temperature took place, without showing any regular curve. The examination of the blood gave negative results.

Conclusion.

Although the strict proof, viz., the demonstration of the piroplasma equi is missing, we are entitled to regard the temperature reaction as due to the inoculation of the blood, since we know from previous experiments that mules support inoculations with immune blood easily.

(F).—Experiments to Show that the Blood of a Mule Immune against Equine Piroplasmosis remains infective for Susceptible Donkeys.

Donkey No. 737, imported from the Argentine to Cape Town and brought directly to Pretoria, was inoculated into the jugular vein on 16th January 1905 with 10 cc. defibrinated blood. A slight disturbance of the temperature took place after fourteen days, and a further disturbance was noticeable after three weeks. There is, however, no proof that the reaction was due to the presence of piroplasma equi.

Donkey No. 741. Same origin as above. It was injected on 15th February 1905 with 10 cc. blood of mule No. 592. On 27th February, viz., twelve days after inoculation, and during a very slight fever reaction, the piroplasma equi was found in the blood. They had disappeared the next day. A second and more distinct reaction started about sixteen days after the inoculation, but the examination of the blood gave negative results.

Conclusion.

The inoculation of blood of a mule immune against equine piroplasmosis produces in donkeys a mild disease, with a primary and a secondary reaction; the inoculation of such blood may therefore be made use of as a practical means of protecting donkeys against the disease.

(*G*).—*Experiments to Show that the Blood of a Donkey Immune against Equine Piroplasmosis is infective for Susceptible Horses.*

Horse No. 600, Argentine, about four years old, brought from board ship in Durban to Pretoria, was on 16th January injected intrajugularly with 10 cc. defibrinated blood of a donkey, No. 306.

This donkey was a country-bred animal, and had been hyperimmunised with horse-sickness blood of horses. On 22nd January 1905 the temperature began to rise and reached 106° F. on 25th January, on which day the piroplasma equi was frequent. A very severe attack of biliary fever had developed, the symptoms of jaundice being well pronounced on the 25th of January. The horse recovered from the first reaction, when a slight second one took place, closely followed by a third distinct one, during which the piroplasma equi was seen again. The horse recovered, but had lost enormously in condition.

Horse No. 723, Argentine, about three years old, brought from board ship in Capetown directly to the laboratories. Inoculated subcutaneously on 15th February 1905 with 10 cc. defibrinated blood of donkey No. 306 (compare previous experiment). On the fifth day after the injection a slight reaction began, and on the ninth day piroplasma equi was present. The number increased during the next few days, rosettes being frequently noticed. The reaction was not exactly a severe one, but notwithstanding this all the clinical symptoms of jaundice had developed. A second slight reaction occurred on 7th and 8th March, viz., three weeks after inoculation, during which, however, no piroplasmata were seen. The horse contracted horse-sickness spontaneously, and died on 15th March.

Conclusions.

(1) The inoculation of blood of a donkey immune against equine piroplasmosis produces this disease when injected into susceptible horses.

(2) In the course of the disease two reactions are noticeable.

(3) Although the clinical symptoms indicated a fully developed disease, it was not so grave as to cause death. There thus exists some prospect of being able to utilise the blood of immune donkeys for inoculation purposes against biliary fever of the horse.

(*H*).—*Experiments to Show whether the Blood of a Donkey Immune against Equine Piroplasmosis will Produce the Disease when injected into Susceptible Mules.*

Mule No. 590, Argentine, about three-and-a-half years old, brought from board ship at Durban to Pretoria, was inoculated into the jugular vein on 16th January 1905 with 10 cc. defibrinated blood of donkey, No. 306. On the ninth, tenth, and eleventh days a slight reaction was noticeable, the temperature being on the 25th January 103·8° F.

Piroplasma was not seen. Nothing to, indicate a second reaction occurred after this.

Mule No. 591, Argentine, about three years old, same origin as previous one. It was inoculated on 15th February. A slight reaction took place, and a second one, likewise slight, was noticeable about the third week. Piroplasma equi was not seen.

Conclusion.

The blood of a donkey immune against equine piroplasmosis produced a slight reaction in susceptible mules, which reaction corresponds in its character to that seen in mules injected with immune blood of horses and mules. There exists every prospect of being able to utilise the blood of an immune donkey for the inoculation of mules against biliary fever.

(*I*).—*Experiments to Show that the Blood of a Donkey Immune against Equine Piroplasmosis will produce the Disease when Injected into Susceptible Donkeys.*

Donkey No. 738, Argentine, brought from board ship at Cape Town to Pretoria, was injected into the jugular vein with 10 cc. defibrinated blood of Donkey No. 306 on 16th January 1905. The ninth day afterwards the reaction commenced, and on the tenth day piroplasma equi was very frequent. It increased in numbers during the following days, and the donkey died on 28th-29th January 1905.

The *post-mortem* was made in the morning. The flesh had a slightly brownish colour, and the blood also had a brown hue. There was a large *ante-mortem* clot in the right, and a red clot in the left, ventricle. There were no petechiæ in the heart. The liver was slightly jaundiced. The spleen was very much enlarged, about four to five times its normal size, and the pulp was very soft. The kidneys were pale, and the bladder was full of red urine.

Donkey No. 744, Argentine, same origin as previous one, was injected subcutaneously on 15th February 1905 with 10 cc. defibrinated blood of Donkey No. 306. A first reaction was noticeable after five days, and piroplasma equi was seen for the first time on 22nd February; it was still present on the 24th, but absent on the following day. Between the first and second reactions there was an interval of four days. The second reaction lasted three days, but no piroplasmata were discoverable during that time.

Conclusion.

The blood of a donkey immune against equine piroplasmosis produced a deadly disease in a susceptible donkey, and a distinct reaction in a second one. There is, accordingly, but little prospect of being able to utilise the blood of an immune donkey for inoculation purposes against piroplasmosis in donkeys.

Summary of Conclusions.

(1) The piroplasma found in the mule and the donkey is identical with the piroplasma equi first found in the horse.

(2) The disease caused by this piroplasma is inoculable with blood of immune animals into susceptible ones belonging to the domesticated species of the genus *equus.*

(3) The horse shows the greatest susceptibility for this piroplasma; the donkey is less, and the mule the least, susceptible.

(4) The possibility of a practicable inoculation against the piroplasmosis stands in the reverse order of the susceptibility. The mule may be safely inoculated with immune blood of any of the three respective equines; the immune horse-blood produces the severest reaction, the immune mule-blood causes little reaction, and so does the immune donkey-blood.

For practical purposes the inoculation of mules with blood of immune donkeys may prove successful.

The donkey proves to be equally susceptible to immune blood taken from horses and donkeys, and shows the slightest reaction to the injection of immune mule-blood.

For practical purposes the inoculation of donkeys with blood of immune mules may prove to be successful.

The horse, which is extremely susceptible to piroplasmosis due to the injection of immune blood from the horse and mule, seems to suffer less from the inoculation of immune donkey-blood.

For practical purposes the inoculation of horses with blood of immune donkeys would have to be taken into consideration.

EPIZOOTIC PNEUMO-ENTERITIS OF THE TURKEY.

By SYDNEY DODD, M.R.C.V.S., Demonstrator of Pathology,
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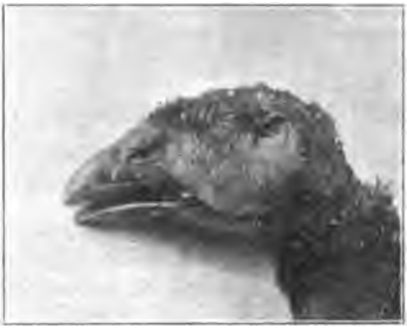
ON the 22nd August last, in the absence of Professor M'Fadyean, I was consulted with regard to a disease amongst turkeys in Norfolk, which was causing great mortality. The history of the outbreak is as follows:—

About five weeks previously the owner, who kept a flock of 120 turkeys, observed some of them to have "swollen heads." About two weeks later acute symptoms set in, several of the birds dying daily, until, out of the flock of 120, only 70 were left, and the deaths still continued. The affected turkeys lost their appetite, and evinced a desire to keep away from the other birds. Feathers became ruffled. Usually a swelling, which varied in size in different birds, developed under one or both eyes. Tongue furred. There was a discharge from the nostrils and mouth. Motions were very loose, sometimes white in colour. Affected birds became very thin.

The birds were never kept shut up, but were allowed to wander over an area of fully 15 acres besides some 10 or 12 acres of wood. Fowls had always been running and feeding with the turkeys, but only the latter were affected in any way. No birds of any kind had been imported from other localities for a considerable time. The owner stated that "swelled head" had been noticed among turkeys on other farms, but he was unable to say whether any deaths had occurred. He had previously written to a weekly agricultural paper, and was informed that the deaths were due to "foul ground," caused by overcrowding.

At my request, three dead turkeys were forwarded on 24th August to the laboratory for *post-mortem* examination, and at the same time two live birds for observation. A sixth, to which I shall refer later on, was sent alive on 7th September. The following are the notes of the *post-mortem* examinations:—

Turkey No. 1.—A nearly full-grown hen. Body rather emaciated. A slight fluctuating swelling under both eyes. On incision, a clear viscid liquid escaped from the swellings. Slight discharge from the nostrils, and a rather profuse discharge from the mouth which had the same character as that obtained from the swelling under the eyes. Ovoid bacteria were present in large numbers in these discharges. Inside of mouth thickly covered with a creamy diphtheritic deposit. Crop contained a quantity of whey-like material, but no food. Heart normal; no visible pericarditis. Blood liquid, and contained a few ovoid bacilli. Right lung quite solid, and on section showed numerous light yellow foci. Large bronchi plugged with yellow fibrinous lymph. Great numbers of ovoid bacilli present in smears taken from the lung. Intestines markedly inflamed and contained a colourless jelly-like material. Spleen and liver normal in appearance. Cultures made from the heart blood and lung gave almost pure growths of the bacillus about to be described.



Head of a diseased turkey, showing a swelling under the eye.

Turkey No. 2.—A young hen. Body thin. A swelling about the size of a walnut below the left eye, having the same characters as in the previous case. Thick discharge from the nostrils, and also from the mouth. A few worms (syngamus trachealis) in the trachea. Heart normal; no evident pericarditis. Apex of left lung hepatised. Two solid masses of yellow fibrinous lymph in the pleural cavity. Slight general enteritis. Liver and spleen normal in appearance. Smears from the swelling under the eye and the fibrinous lymph showed large numbers of ovoid bacilli, but they were very scanty in the heart blood.

Turkey No. 3.—A young bird. Body well nourished. Only a slight swelling under the eyes. Tongue furred. A viscid clear liquid dribbling from the mouth. Heart normal; no evident pericarditis. Both lungs hepatised, and on section show a number of light yellow foci. Spleen and liver normal. Intestines slightly congested in parts. Smears from the lung revealed ovoid bacilli in great numbers. In the heart blood they were fairly numerous. Cultures from the blood and lungs gave pure growths of the bacillus.

Turkey No. 4.—A nearly full-grown hen. Arrived alive at the laboratory at 5 P.M., 24th August. Bird very ill and unable to stand. Died at 8 A.M., 25th August. *Post-mortem* made at 10 A.M. Body rather thin. A fluctuating swelling under the right eye, about the size of a Tangerine orange, containing a clear viscid liquid. Inside and at the base of the swelling, but distinct from it, was a small abscess with light yellow, almost solid contents. Both the clear liquid and the abscess swarmed with ovoid bacteria. A profuse discharge from the nostrils and mouth; inside of mouth lined with a creamy diphtheritic deposit. A few worms (three pairs) in the trachea. Heart normal; no pericarditis. Blood contained a few bacilli. The bases of both lungs appeared to be occupied by masses of yellow fibrinous lymph, which as usual swarmed with ovoid bacilli. Spleen and liver normal. Crop contained about 2 ozs. of liquid, in which were floating masses of cheesy-looking material. Whole of intestines congested. Cultures from the heart blood gave pure primary growths of the bacillus, whilst those made from the fibrinous lymph and swelling on the head also showed a few accidental organisms.

Turkey No. 5.—A half-grown bird sent alive on 24th August for observation. Bird very weak and thin on arrival. Both sides of head under the eyes much swollen. Slight discharge from nostrils and mouth. Gaping continually (due to syngamus trachealis). Marked diarrhœa.

30th August.—Bird very ill. Feathers ruffled. Profuse diarrhœa. Discharge from nostrils more marked. Refuses food.

4th September.—Bird much better, and eating a little. Swellings on head decreasing.

16th September.—Still improving, eating well. Diarrhœa slight. Swellings markedly less.

Turkey No. 6.—This bird, a young one, was sent alive to the laboratory on 7th September. The owner, a medical man, had observed that some of the birds had "swollen heads" but developed no acute symptoms of disease, and apparently recovered without ever having been very ill. He was therefore of opinion that the birds were suffering from two diseases, the one of which "swelled head" was a symptom being quite distinct from that affecting the lungs and causing death. I therefore requested him to send me a bird which he considered was affected with "swelled head" only, and showed no lung or intestinal trouble. I may add that, at my suggestion, the whole of the remaining turkeys were inspected by him twice a day in order to detect any fresh cases. The turkey appeared fairly lively on arrival, but was in poor condition. There were marked swellings under both eyes, a profuse discharge from the nostrils, and the tongue was thickly furred. There was also evidence of slight diarrhœa. The bird was kept under observation for five days, during which time it had a good appetite and showed no urgent symptoms. The nasal discharge continued, but diarrhœa was not very marked. It was killed by chloroform on 12th September.

Post-mortem.—Body thin. Both swellings under the eyes contained the usual colourless viscid fluid, in which were large numbers of ovoid bacilli. Lungs apparently normal, but cover-glass preparations showed numbers of bacilli like those of the previous cases. Heart normal; no pericarditis. No bacilli to be seen in heart blood. Liver and spleen normal. Intestines showed numerous ecchymoses. Small intestines contained a number of round worms.

Apparently this bird was suffering from a mild attack of pneumo-enteritis. That an attack of the disease is not necessarily fatal is shown by the fact that turkey No. 5 was badly affected on arrival at the laboratory, but is now (16th September) on the road to recovery.

On my communicating with the owner, describing the lesions observed by me, he made *post-mortem* examinations himself, and wrote:—"I have examined quite a dozen birds. The pleura and lungs have been full of a cheesy purulent material. In one or two cases hardly any lung tissue has been found remaining. No pericarditis has been seen. The birds are in very thin condition."

The Causal Organism.—The organism found in the swellings on the head, the lungs, fibrinous lymph, and heart blood has been identical in all cases, morphologically and culturally, and I consider it to be responsible for the deaths of the turkeys in this outbreak, although the disease was not experimentally transmitted to turkeys owing to want of subjects and time. The organism is a short ovoid bacillus belonging to what has been termed the fowl-cholera type, and, whether in the blood, tissues, or artificial cultures, cannot be distinguished from the bacillus of fowl cholera. Mixed with the ovoid forms there are always a few that are more distinctly rod-shaped, thus giving at first sight the impression of impurity in the preparation. It stains with aqueous solution of any of the aniline dyes, but not by the method of Gram, Gram-Weigert, or Claudius. With aqueous solution of methylene-blue the bacilli exhibit bi-polar staining, the two poles being deeply stained, whilst the centre remains unstained, thus often looking like diplococci. Leishman's method of staining shows the morphology of the organism to the best advantage, especially in blood films. The poles stain deeply, and also what appears like a thin limiting membrane around the bacillus, thus rendering the unstained portion more prominent. The bacillus is non-motile.

Cultural Characters.—On slanting agar at 37° C. growth is visible in twenty-four hours. If the colonies are discrete, they are circular and translucent. If the inoculating material is rich in bacilli, the entire inoculated surface of the agar is covered with a thin translucent pellicle, which darkens with age. In bouillon at body temperature the medium becomes uniformly turbid in twenty-four hours, but with continued incubation a flocculent deposit collects at the bottom of the tube. In stab cultures in 10 per cent. gelatine kept at room temperature a growth is visible along the needle track in forty-eight hours. No lateral outgrowths develop. The gelatine is not liquefied. On potato incubated at 37° C., provided the inoculating material is rich in bacilli, a faint waxy-looking growth appears after a few days, but does not tend to spread.

Experimental Inoculations.

Fowl No. 1. 28th August 1905.—Inoculated into the pectoral muscles a full-grown hen with half an agar culture of the bacillus obtained from the heart blood of turkey No. 1. The slanting agar culture was incubated at 37° C. for forty-eight hours, then about 2 cc.

of sterile water were poured into the tube, and the growth detached and emulsified by means of a sterile platinum needle. During the following three or four days the fowl was dull and not feeding well. Since then it became more lively, its appetite returned, and it is now (16th September) apparently quite well.

Fowl No. 2. 28th August.—Inoculated into the pectoral muscles an adult hen with half an agar culture of the bacillus isolated from the fibrinous lymph of turkey No. 4, incubated at 37° C. for forty-eight hours. The emulsion was made as described above. On the following two days the bird was moping, but it improved after that, and at the time of writing (16th September) appears quite well.

Rabbit No. 1. 25th August 1905, 3 P.M.—Inoculated a full-grown rabbit subcutaneously with about four drops of an emulsion in bouillon made from the fibrinous lymph from the lung of turkey No. 4, a piece of fibrinous lymph about the size of a split pea being teased out by means of sterile needles, and emulsified with about 3 cc. of bouillon.

26th August.—Rabbit found dead at 6 A.M. (fifteen hours after inoculation).

Post-mortem.—An area of inflammatory œdema about the size of a florin around the seat of inoculation. Axillary lymphatic glands swollen and deeply congested. Inguinal glands slightly ditto. Spleen and kidneys normal. Liver congested. Pericardial sac contained a considerable amount of a clear fluid. A few ecchymoses on endocardium. Blood swarming with bipolar-staining bacilli. Lungs and intestines normal. The bacillus was recovered in pure culture on agar from the heart blood.

Rabbit No. 2. 4th September.—Mixed a bouillon culture incubated at 37° C. for forty-eight hours, of the bacillus from the heart blood of rabbit No. 1, with a handful of bran, and fed it to an adult rabbit. More than half of the mixture was eaten within half an hour, but the result was entirely negative, the rabbit being alive and quite well at the present (16th September).

Guinea-pig No. 1. 25th August 1905.—Inoculated a guinea-pig subcutaneously with 1 cc. of an emulsion in bouillon of fibrinous lymph from the lung of turkey No. 4 (four drops of an emulsion of the same material killed a rabbit in fifteen hours). A suppurating lesion appeared at the point of inoculation, with extensive œdema of the surrounding tissue. The animal showed no systemic disturbance, and on 16th September the œdema was disappearing and the ulcer healing up.

Guinea-pig No. 2. 1st September 1905.—Inoculated an adult guinea-pig subcutaneously with 1 cc. of a bouillon culture, incubated for forty-eight hours at 37° C. of the bacillus from the heart blood of pigeon No. 2. The result was practically the same as with guinea-pig No. 1, except that the suppuration and œdema was much less marked, and by 16th September had nearly disappeared.

Guinea-pig No. 3. 1st September.—Inoculated subcutaneously an adult guinea-pig with 1 cc. of a bouillon culture, incubated at 37° C. for forty-eight hours, of the bacillus from the fibrinous lymph of the lung of pigeon No. 2. The result was just the same as with guinea-pig No. 2.

Pigeon No. 1. 28th August.—Inoculated a pigeon into the pectoral muscles with half an agar culture, incubated at 37° C. for forty-eight hours, of the bacillus obtained from the heart blood of rabbit No. 1, an emulsion being made from the slanting agar in the manner previously described.

29th August.—Pigeon appears quite well. On the 2nd September and for several days following the pigeon was very dull. 6th September, pigeon feeding well and quite lively. 16th September, pigeon apparently quite well.

Pigeon No. 2. 28th August.—Inoculated into the pectoral muscles a pigeon with half an agar culture incubated for forty-eight hours at 37° C., of the bacillus obtained from the heart blood of turkey No. 4. The emulsion was made as before.

29th August—Pigeon appears well. 30th August, very dull. 31st August, pigeon found dead at 8 A.M.

Post-mortem.—An area of dry necrosis about the size of a walnut around the seat of inoculation and extending along the needle track. A clear viscid fluid escaping from the mouth. Inside of mouth covered with a diphtheritic deposit. Apices of lungs hepatised. Large bronchi plugged with yellow fibrinous lymph, cover-glass smears from which showed bipolar-staining bacilli in enormous numbers. No evident pericarditis. Heart blood contains only a few bacilli. Spleen and liver normal. Patches of intense inflammatory congestion scattered along the whole of the intestines. Contents jelly-like. The bacillus was recovered in pure culture on agar from the lungs and heart blood.

Pigeon No. 3. 4th September 1905.—Inoculated into the pectoral muscles a pigeon with 1 cc. of a bouillon culture, incubated for forty-eight hours at 37° C., of the bacillus obtained from the heart blood of rabbit No. 1.

7th September—Pigeon found dead. Had been moping since 5th September.

Post-mortem.—Diphtheritic deposit lining mouth. An area of dry necrosis about the size of a shilling at the seat of inoculation. No evident pericarditis. Very few bacilli in heart blood. Lungs deeply congested. No hepatisation. Large numbers of ovoid bacilli obtained in smears from the lung tissue. Liver contained an area of dry necrosis about the size of a sixpence, containing numbers of the usual bacilli. Spleen normal. Intestines moderately congested. The bacillus was recovered in pure culture from the lungs and heart blood.

Unfortunately further experiments could not be carried out owing to my leaving to take up an appointment in South Africa.

About fourteen years ago (*Journal of Comp. Path. and Therap.*, Vol. VI., December 1893), Professor M'Fadyean investigated an outbreak among turkeys at Newport Pagnell, and he described the disease under the heading of "Epizoötic pneumo-pericarditis of the Turkey."[1] He also identified and described as the causal organism a bacillus resembling that of fowl cholera, but he considered that the bacillus found in the cases of pneumo-pericarditis was not identical with the fowl cholera organism.

His reasons were (1) The disease was almost confined to turkeys although fowls were allowed to mix with them (only one fowl died in the outbreak). (2) Pneumonia was a constant lesion. (3) The turkey bacterium was only feebly pathogenic for the fowl, whereas the bacillus of fowl cholera is certainly and rapidly fatal to that species by inoculation or ingestion. (4) The bacillus of fowl cholera is invariably fatal, generally within twenty-four hours, when inoculated into the pigeon; but with the turkey bacterium the inoculation was either without apparent effect, or death did not occur until some considerable time after inoculation. (5) The fowl cholera bacillus is very fatal to the rabbit if administered by the alimentary canal. One feeding experiment with the turkey bacterium was negative.

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[1] **I am indebted to Professor Macqueen for calling my attention to this article.**

On clinical and experimental grounds, the disease described by Professor M'Fadyean and the one now under notice have much in common, but there are several points in which they differ that require mentioning:—

(1) The organism described by Professor M'Fadyean was motile. The one under present notice is non-motile.

(2) "Swelled head" was almost a constant feature in the present outbreak. Professor M'Fadyean does not mention it in his account, so presumably it was absent.

(3) In none of the cases examined by me (five) or by the owner (twelve) was there any evidence of pericarditis. It was an important lesion in the 1893 outbreak.

(4) Enteritis was a constant feature in the present cases. In Professor M'Fadyean's no enteritis was observed in the naturally occurring ones, and it was seen in only two of the experimental turkeys.

Reviews.

A Manual of Veterinary Hygiene. By Col. F. Smith, C.M.G., Army Veterinary Department. Third Edition. Rewritten. London: Baillière, Tindall & Cox, 1905.

During the eighteen years which have elapsed since its first appearance British veterinary students have been familiar with Col. Smith's text-book of Veterinary Hygiene, and both teachers and students have found in it a valuable aid in the study of a branch of veterinary education which is daily acquiring greater importance. The new edition is practically a new work, and therefore deserves more than a passing notice. The first feature of the new edition which impresses the reviewer is its greatly increased size, the amount of matter contained in it being nearly double that of its predecessors. This is due in part to the fact that most subjects are treated more exhaustively and thoroughly in this edition, but mainly to the inclusion of matters of hygiene which are now dealt with for the first time.

The opening chapters are devoted to water, air, ventilation, food, and buildings, and constitute about one-third of the work. Succeeding these are chapters which severally deal with soils, disinfection, parasitic and microbic diseases, State hygiene, and municipal hygiene, while the remainder of the work treats of the care and management of animals, the hygiene of breeding, the transport of animals by sea and land, the hygiene of animals employed in war, climate, and statistical inquiry. This bare enumeration of the headings under which the matter is arranged will prevent any surprise when it is said the work now runs to over 1000 pages.

Needless to say, no single individual could pretend to write with authority on all these aspects of hygiene in the widest sense of the word, but Col. Smith, in writing several of the chapters, has been fortunate enough to secure the co-operation of a number of eminent members of the profession, and he has at the same time made judicious use of recent contributions to the literature of veterinary hygiene which have appeared in the professional journals and elsewhere. He has thus succeeded in producing a work in which every division of the subject is dealt with more or less authoritatively. Even the experienced practitioner will learn much from it that is applicable to his everyday work It will perhaps be still more useful to those members of the profession who have to act as veterinary inspectors under the Contagious Diseases of Animals Act, and students will find in it an indispensable guide in preparing for their examinations.

The book is freely illustrated, and the work of the publishers has been well done.

Speziełle Pathologie und Therapie der Hausthiere. Von Dr Franz Hutyra, Professor der Seuchenlehre, und Dr Josef Marek, Professor der spez. Pathologie und Therapie, an der Tierärztlichen Hochschule in Budapest. 1 Band. Jena: Gustav Fischer, 1905.

THE basis of this work is the text-book with the same title which was first published in the Hungarian language by Professor Hutyra. In preparing the second edition Professor Hutyra and his colleague, Professor Marek, have co-operated, the former being responsible for the Infectious Diseases, and the latter for the Diseases of Organs. The new edition, which was practically a new work, much larger than the first, was published in Hungarian last year, and its favourable reception by the veterinary profession in Hungary has induced the authors to undertake the translation of the work into German.

The first part, which has now been issued, is a bulky volume of 873 royal octavo pages, and in it are dealt with the infectious diseases, diseases of the blood and of blood formation, diseases of the spleen, diseases due to disturbed metabolism, and diseases of the circulatory system.

The work is one that naturally invites comparison with the well-known text-book of Friedberger and Fröhner, or, so far as the infectious diseases are concerned, with the equally well-known treatise by Nocard and Leclainche. That it is one of high merit will be inferred when we say that it will bear comparison with either of the standard text-books just mentioned. As compared with the former of these, it has the advantage of being somewhat stronger on the pathological side, and over the latter it has for the moment the advantage of being more perfectly up-to-date, though in some of the sections (notably the one dealing with trypanosoma diseases), the information given is less complete than in the French treatise. There are 132 illustrations in the text, and three coloured plates at the end of the volume.

Putting comparisons aside, however, it may be said that the work is a most valuable addition to veterinary literature, and that it reflects great credit on its authors, alike for their learning and their industry.

Cattle Tuberculosis. A Practical Guide to the Agriculturist and Inspector. By Harold Sessions. F.R.C.V.S., etc. Second Edition. London: Baillière, Tindall & Cox, 1905.

It is to be hoped that the appearance of a second edition of this little work justifies the conclusion that the first one found a market among the class for whom it was mainly intended, viz., British stock-owners, who certainly still stand in much need of enlightenment with regard to the cause of tuberculosis among farm animals, and the private and public measures that ought to be opposed to it. In language which is as far as possible non-technical, Mr Sessions has given a very useful *exposé* of the pathology, symptoms, and prophylaxis of bovine tuberculosis. Naturally, the tuberculin test, and the regulations which in this and other countries have been put into force with the object of checking the spread of the disease among cattle, and preventing its extension from cattle to human beings through the meat and milk of tuberculous animals, come in for special attention, and some useful information is also given regarding the insurance of cattle against seizure on account of tuberculosis or other diseases after slaughter. We can strongly recommend the book to farmers and dairymen who desire to know how they may best proceed in order to prevent or minimise the loss which uncontrolled tuberculosis is capable of causing in their herds.

Report on Dourine in Different Breeds of Equines, together with an Account of Vesicular Exanthema and Piroplasmosis which occurred as Complications. By Alfred Lingard, M.B., M.S., D.P.H., Imperial Bacteriologist to the Government of India. Calcutta: Office of the Superintendent of Government Printing, India, 1905.

Dr Lingard has during the last few years had exceptional opportunities to study dourine—a disease which, however it may have been introduced into India, is now far from rare, and threatens to seriously interfere with horse-breeding in that country. The account of his observations which is given in this monograph, although not adding anything of importance to what was previously known on the subject, is interesting, and the detailed history of some typical cases, supplemented by a number of excellent photographs, will be found of real value by those who have not had previous experience with the disease. The same may be said of Dr Lingard's account of the cases of vesicular exanthema, which, it appears, has in the past been frequently confounded with dourine. The part of the monograph, however, which is of greatest interest is that devoted to piroplasmosis, for it indicates that a disease (or diseases) of that kind is prevalent in India to a degree that has not hitherto been suspected. The author has found piroplasmata in the blood of horses, cattle, buffaloes, camels, elephants, deer, goats, sheep, dogs, cats, monkeys, and human beings, and, what is most remarkable of all, he has succeeded in transmitting the disease by inoculation to rabbits, guinea-pigs, and rats. Unfortunately the details of these cases are lacking, but a full account of the life history of the piroplasms and of their connection with the ixodes, culicidæ and diptera generally, together with the symptoms produced in the host, is promised as a separate publication. This will be looked forward to with interest.

CLINICAL ARTICLES.

FRACTURE OF THE SUPERIOR MAXILLARY BONE IN A HORSE.

By G. H. LIVESEY, M.R.C.V.S., Hove.

THIS patient, a hunter, first seen on 15th July, had just been brought up from grass, where he had been turned out for about two months. Some three or four weeks before my seeing him he had been kicked by another horse in the face, and the skin broken ; he had rubbed the sore thus formed on a wooden fence, probably owing to irritation by flies. The wound had been washed and allowed to heal slowly. His condition gave no cause for anxiety until the submaxillary glands were noticed to be enlarged, and he was found to have several patches of ringworm on his skin. He was therefore taken up and my services were requisitioned.

State on Examination. The frontal bone on the right side was at once seen to be depressed, and the side of the face below the right eye somewhat swollen. Over the antero-inferior portion of the inferior maxillary sinus was a small fistula with granulating edges, from which there issued a very slight and thin discharge. An intermittent mucopurulent discharge was also coming from the right nostril, and both submaxillary glands were enlarged to about the size of two pigeon's eggs. There was a scar of another wound about two inches below the right eye. Several patches of ringworm were noticed about the body and legs, but otherwise the horse was in very good condition. I could detect no fracture of the bones of the face on pressing with the finger, all the tissues beneath the skin being swollen and indurated. Probing the fistula gave unsatisfactory results, as its course was somewhat sinuous.

I explained to the owner that the maxillary bone was probably broken, but had great difficulty in getting him to believe that my view was correct. He, however, eventually sent the horse to me for treatment, and on 20th July I operated as follows: The edges of the fistula were cut away, when it was seen that the wound penetrated the levator labii superioris muscle and exposed the bone, that there was a depressed fracture of the maxillary bone, and that the sinus contained a quantity cf granulation tissue bathed in mucopus. The opening was then enlarged, and the edges of the bone pared. From the cavity were then removed with long fine forceps, at distances varying from two to five-and-a-half inches from the surface wound, eleven or twelve splinters of wood about the thickness of a safety match, and varying in length from a halt to two inches. Five inches from the surface a loose triangular plate of bone, whose longest side measured three quarters of an inch, was found and removed, allowing exit to a quantity of purulent material. The wound was well syringed out, but the discharge seemed interminable ; so, in order to get better drainage, I made an opening through the wall of the sinus into the nose. The bleeding was profuse for a few minutes, and with the blood came a quantity of pus. I next trephined the frontal sinus and found it free from pus, but there was a considerable swelling behind the scar mentioned above, and I have no doubt that this was the seat of a subcutaneous fracture where healing had taken place without interference. Both sinuses were thoroughly washed with a gentle stream from a hose pipe, the force of water being sufficient to thoroughly clear the nose of all blood clots and discharges. The wounds were plugged and the horse left for the night.

During the evening about five or six ounces of blood-stained pus came from the wound, and I was not satisfied with the means of drainage provided. Cleaning out the cavity of the wound, the inferior maxillary sinus seemed completely filled with granulation tissue, in the midst of which pus had burrowed in all directions. There had been no further discharge from the nose, so I inferred that the nasal opening had become occluded. I decided to allow it to heal and to trephine below the external wound. The point of the trephine was inserted about an inch anterior to the inferior extremity of the zygomatic process, and the same distance above it. The aperture thus made opened on to the septum, exposing both superior and inferior sinuses, from which three or four ounces of blood-stained pus at once escaped, and a further quantity when the wound was flushed with water. The new tissue in the lower sinus was thoroughly broken down between the original wound and that just made, and all pus carefully washed out, after which all three sinuses were irrigated with carbolic solution and the frontal and maxillary sinus openings plugged with corks, that in the latter having a hole in its centre to allow of the discharge draining freely away. The patient's face was washed, dried, and well coated with corbolised vaseline.

On the 25th the opening into the frontal sinus was allowed to heal, the granulation in the lower wounds removed with the knife, and a gauze drain inserted. Twice every day the wounds were well syringed out with either chinosol or zinc chloride solution. On the 28th the gauze drain was removed and the original wound allowed to heal, only the lower opening to the inferior maxillary sinus being kept open and dressed daily. On the 31st all the wounds except the latter were practically healed, and the nasal discharge of no consequence, the horse feeding well and all swelling of the face gone. The lower opening was completely healed on 8th August, and the patient discharged from hospital cured.

OPERATION ON THE NOSE IN A DOG.

By the Same.

ABOUT a year ago I was consulted about a Retreiver dog, aged about ten years, which was reported to be suffering from an ordinary cold in the nose. In spite of treatment the discharge from the nostrils continued more or less throughout the winter. In January the dog was brought to me with a history of severe intermittent bleeding from the right nostril. I kept him for a week and failed to find any obvious cause, so advised his being kept very quiet and carefully dieted. In June he was reported to be much worse again, and in July he came to me once more, and I had the pleasure of showing him at the meeting of the S. C. V. S. on the 7th. My diagnosis of his case was that of polypus, and I had already made up my mind to make an exploratory opening in the nostril, and also into the frontal sinus in case of there being a pentastome in the latter situation.

On 14th July I trephined the skull and nostril in the following manner. The forehead and nostril on the right side were shaved and thoroughly prepared, and the dog then put under chloroform. When completely anæsthetised a probe was used to test the patency of each nostril, and to find if possible the situation of the suspected growth. As I could, however, gain no information from this, I made an incision from the middle line of the nose immediately above the nasal branch of the angular vein, continuing it downwards and backwards below the level of the lower eyelid, and just behind the dental foramen, thus escaping the larger bloodvessels, to meet another incision starting from midway between the inner canthus of the eye and the middle line of the nose. The triangular flap of skin was dissected up and held back, and the bone exposed. All hæmorrhage was at once stopped by use of solution (1-2000) of adrenalin (takamine). The bone was then removed with a half-inch trephine and the nasal cavity exposed. Hæmorrhage, which threatened to be profuse, was once more checked by adrenalin, and the head was kept in a dependent position for fear of fluids entering the larynx by the naso-pharynx, the opening of which was immediately below the operation wound. After careful exploration all I could find was a tumefied condition of the lining membrane, which in two places seemed to resemble small mucous polypi. I removed these, and enlarged the opening a little higher, so as to more fully expose the turbinated bones. Beyond being covered with a very thick mucus, these appeared normal. I introduced a dental mirror into the cavity and examined the walls, but could find no trace of new growths or of any ulceration. The condition of the patient remaining very good, I decided to open the frontal sinus. This was done by making only one incision in the skin about half an inch from the middle line and parallel with it. The lips of the wound were then drawn apart and the bone exposed. The trephine was placed in the centre of this wound, with the centre piece on a line joining the highest points of the two orbital rims. The sinus was found to contain a quantity of mucus but no pentastome; the membrane, however, showed pink spots like small granulations. As the dog had now been under chloroform quite long enough, I syringed out the sinus and nostril, removing all small blood clots, and, as the dog recovered consciousness, tested the patency of the naso-pharynx. This appeared quite free, but the nares seemed more or less occluded and respiration was to a great extent performed through the operation wound. The sinus was cleaned out with sterile swabs and then flushed out with a 1-1500 solution of perchloride. The skin wound over the frontal sinus was left untouched, but the triangular flap was stitched with silkworm gut; dried carefully, and sealed with "New Skin."

The patient had been under chloroform for about an hour and twenty minutes, but made an excellent recovery. The administration of the chloroform was rather difficult, as the use of an inhaler interfered with the site of the operation, but this was overcome by using a Junker (metal) inhaler, and only just inserting the end of the nose inside the rubber at the end. The preparation "New Skin," which I used to cover the wound, I have found vastly superior to all collodion preparations, as it gives a much thicker covering, dries quicker, and keeps the wound quite aseptic, and animals do not interfere with wounds protected by it.

From this date onward the wounds healed without interruption, and the nose bleeding entirely ceased. There was always a little discharge from the nose, best seen in the morning, but I could find very little interference with normal respiration. The dog was discharged from hospital on 31st July.

A CASE OF HYDRONEPHROSIS IN A COW.

By A. SPREULL, F.R.C.V.S., Dundee.

On 21st June last I was called to see an Irish-bred, eight or nine-years-old, shorthorn-cross, milch cow, the property of a dairy farmer in this neighbourhood. On arrival I found my patient looking rather dejected, suffering from profuse fœtid diarrhœa, which the owner informed me had been continuous for a considerable time, but the exact date of its existence could not be ascertained. The skin was dirty and uræmic looking, more especially along the region of the back and the upper parts of the shoulders, the coat staring, and accompanied by a considerable amount of itching throughout this region. The pulse, breathing, and temperature were practically normal, the salient symptoms of the case being, as already stated, the profuse fœtid diarrhœa and the unhealthy itching coat. The appetite, the owner informed me, was very capricious; some days she would eat little or nothing, while other days she would eat anything she was offered, and at such times looked as if there was little or nothing the matter with her. The owner further informed me that he had bought her about three months previously, and that she had continued during the greater part of that time more or less in this condition, but if anything getting rather worse than better, and hence his appeal for my help.

Without pretending to accurately diagnose the case, I proceeded to prescribe for her, gave astringent antiseptics to assist in regulating the state of the bowels, and put her on hay, cotton cake, and crushed grain as food, with a skin dressing of whale oil and spirits of tar, to be followed after a few days with a good wash with warm water in which a sufficient admixture of Jeyes' fluid had been placed, to rid the hide of the dirty, greasy, matted material there accumulated. For a week or two she seemed to improve somewhat, but all of a sudden one day when the owner entered the byre to see her he found her, as he described it, in a dying condition. He there and then promptly had her killed and sent to the inspection department of the public slaughter-house, where she next came under my observation. Here the carcase was found to be well nourished, fairly fat, weighing thirty-four stones after removal of the kidneys, and

THE IMPORTANCE OF BACTERIOLOGICAL EXAMINATION OF THE BLOOD AFTER DEATH.

It was formerly thought that *post-mortem* bacteriological examination of the blood might be of very great assistance in determining the cause of death, and, in fact, the specific microbes of several infectious diseases have been discovered by examination of the cadaver. Bacteriological examination of the blood after death is undertaken with the object of discovering the organisms which are present and the part they play in the production of death. It aims therefore at recognising amongst the bacteria present those which have entered the blood during the period of agony and after death. Many authors believe that during the last moments of life and immediately after death the body is invaded by different putrefactive micro-organisms, and that as a consequence the presence of a bacterium in the blood of the heart is no proof that it has caused death.

A lively controversy has recently arisen on this point between Drs Simmonds of Hamburg and Canon of Berlin. Dr Simmonds, in an exhaustive study to be found in *Virchow's Archiv für Pathologische Anatomie und Physiologie*, page 175, No. 3, 1904, gives the results of his bacteriological researches on the cardiac blood of 1200 dead bodies.

He concludes that important facts can be deduced from systematic bacteriological examination of the blood of the dead body. His principal experiments consisted in making cultures from the cardiac blood. He suggests that the microbes whose existence he has proved in the heart blood are the specific microbes that can be found free in the circulating blood during life, and that they have not arrived in the heart from other parts of the body after death. In these investigations the autopsies were made twelve to thirty-six hours after death and in some cases even later. The investigator further claims to recognise the different microbes due to secondary invasion, despite the fact that in 95 per cent. of these attempts only one variety was found, namely streptococcus.

The real causes of death of subjects whose blood was found to contain streptococci were: in 88 cases scarlatina, in 38 cases diphtheria, in 28 cases phthisis, in 58 cases erysipelas, in 29 cases suppurative cellulitis or phlebitis, in 38 cases pyæmia, septicæmia, or malignant endocarditis, and finally various infectious diseases. Dr Simmonds declares that, except where complications caused death, the results of examination of the blood were negative in chronic alcoholism, leukæmia, pernicious anæmia, diabetes, senile marasmus, and in chronic disease of the nervous system and circulatory apparatus.

Dr Canon, in various publications, the last of which appeared in the *Centralblatt für Allgemeine Pathologie und Pathologische Anatomie*, XV., No. 4, 1904, casts doubt on the correctness of this theory. The principal criticism he makes is that Dr Simmonds examined the heart blood instead of the blood of the peripheral veins, such as the vena media basilica. Dr Canon suggests that two cultures made simultaneously, one with blood from a peripheral vein, the other with heart blood, would in certain cases show great differences, that is, that no microbes would be found in the venous blood, and that a great number would be present in the heart blood. According to Dr Canon these bacteria, in almost every case, have migrated from neighbouring organs, particularly the lungs.

The fact that Dr Simmonds has so frequently found streptococci in the heart blood alone in cases of tuberculosis of the lung seems to furnish the principal argument of his opponent, for it is known that the lungs of phthisical subjects contain many varieties of bacteria which during the final agony or after death slaughterer's, and was said to exhibit signs of glanders. The period of forty-five days covered by the guarantee not having expired, the seller would, in case of glanders, have had to bear the loss. The horse had died from rupture of the liver, and was said by the buyer's veterinary surgeon to show sufficiently clear pulmonary lesions to put the diagnosis of glanders beyond doubt. The lung alone had been examined by the latter. The trachea, larynx, turbinated bones, and frontal sinuses had not been exposed.

The pulmonary lesions were as follows: The parenchyma of both lobes exhibited at various points small nodules with fibrous walls and caseous or calcified contents which could easily be detached from their surroundings. The lesions varied in size between that of a pea and a grain of millet. On passing the hand over the surface of the lung these nodules conveyed the sensation of shot embedded in the parenchyma. After examining the lesions, Messrs Breton and Cheneau opened the trachea, larynx, and frontal sinuses and exposed the nasal septum and turbinated bones, all of which were healthy. Only the pulmonary lesions therefore were suspicious. They therefore concluded that the pulmonary nodules were unquestionably of a parasitic nature. But the buyer's veterinary surgeon, the sanitary veterinary inspector belonging to the buyer's Department, and the inspector of the slaughter-house had unhesitatingly concluded that the disease was glanders from a simple examination of the lung, without pushing their investigations any further.

The sanitary veterinary surgeon of the seller's Department after examining the carcase rejected the idea of glanders. The morbid specimens were then sent to a research laboratory, where they were regarded as non-glanderous. If, therefore, it is easy to distinguish parasitic nodules from non-parasitic nodules, it is also possible to confuse them. Nocard has well described the characters of the two lesions—the parasitic nodule has a well-defined wall; its contents are caseous or calcified, and it is easily enucleated. The fibrous wall of the glanderous nodule merges into the neighbouring parenchyma; its contents are difficult to enucleate. Nocard and Leclainche, in the first edition of the *Traité des Maladies Microbiennes des Animaux*, page 756, say, "Recent parasitic nodules may exactly simulate the grey nodule of glanders. . . . In some cases one finds in the lung a few grey or greyish-white centres which have arrived at similar degrees of development and the diagnosis is doubtful. It is even more difficult to prove the existence of glanders from the examination of only a fragment of lung containing a few centres, apart from a histological and micro-biological examination of the lesions." The veterinary surgeon representing the buyer made a deposition of glanders. His Departmental veterinary surgeon, having recognised glanders at the horse slaughterer's, issued a warrant of infection and set in movement the law, which is always troublesome and costly. No similar measures were taken against the seller. The latter had several horses in his stables, but the sanitary veterinary surgeon had concluded that the disease was due to parasitic lesions. There was then an absolute contradiction between the views of the two sanitary veterinary surgeons. The matter was submitted to arbitration. The arbitrator very happily found a ground for conciliation by causing each party to bear one half of the loss; an unfair solution which injured the seller.

One should always be very reserved and careful when, in examining the lungs of a horse one finds nodular lesions which appear to suggest the existence of glanders. The authors hold that if the turbinated bones, larynx, and trachea show no ulcers or cicatrices the diagnosis of glanders should only be arrived at after the specific bacilli have been discovered either with the microscope or by cultivation, and after inoculating guinea pigs from the suspected lesions and producing the characteristic orchitis. (MM. Breton and Cheneau, *Rec. de Méd. Vét.*, 15th Feb. 1905, p. 81.)

syringe. One to thirty drops of this fluid was introduced into the liquefied agar-agar, well shaken up, and the mixture was poured into sterilised Petri glasses for cultivation.

The *post-mortem* examinations, details of which are given below, were in certain cases made less than two hours after death. The causes of the death in the several cases were as follows:—

In seven cases bullet wounds (three in the chest, two in the abdomen, two in the head).

In one case fracture of the frontal bone with cerebral hæmorrhage.

In two cases fracture of the base of the cranium.

In ten cases disease of the valves of the heart.

In two cases rupture of an aortic aneurism.

In one case poisoning by oxalic acid.

In one case pyæmia resulting from accident.

In four cases lober pneumonia.

In one case peritonitis resulting from perforation of the intestine from a knife wound.

In one case complicated fracture of the skull.

In one case cancer of the breast and liver.

In two cases purulent peritonitis following criminal abortion.

In two cases traumatic pneumonia caused by fracture of the ribs.

In one case a bullet wound in the thigh, amputation and septicæmia.

In one case a still-birth.

In three cases fatty degeneration of the heart.

In one case poisoning by morphia.

In one case cerebral meningitis.

In one case peritonitis following perforation of a typhoid ulcer.

In six cases nephritis.

In seven cases bullet wounds causing almost instantaneous death.

In these different cases Gradwohl detected the presence of bacteria under the following circumstances.

An examination of the annexed table will show that by cultivating with the heart blood positive results were obtained in thirty-nine cases and negative results in eleven, out of a total of fifty experiments. In other words, bacteria were found in the heart blood in 78 per cent. of cases, although it was evident that they were not present there during life. On the other hand, microbes were generally absent from cultures of the blood of the arm vein except in some cases where general infection had occurred prior to death. Under these circumstances the same species of bacteria which had been found in the pus of the infected part were found in the venous blood of the arm. The constant negative results of examination of blood from the vena media basilica clearly shows that in that region there is no migration of microbes after death. Similarly the almost constant presence of streptococci and bacillus coli communis in the heart-blood indicates invasion of the heart during the last moments of life by bacilli from neighbouring organs, that is, the lungs, liver, and intestines. As a consequence the indications obtained from bacteriological examination of the heart-blood after death cannot unreservedly be accepted.

Gradwohl thinks that the most reliable information after death is to be obtained from bacteriological examination of the blood of the vena media basilica. For example, Canon has often repeated that in medico-legal cases the pathologist is unable to find the determining cause of death. All the vital organs seem normal. A bacteriological examination of blood from the arm will reveal an infection due to streptococci which leaves no mark visible to the naked eye in the principal organs.—(Dr R. B. H. Gradwohl, *Ann. de l'Inst. Pasteur*, p. 767, Vol. XVIII., No. 12, 25th Dec. 1904).

No.	*Cause of Death.*	*Time between Death and Autopsy. Hours.*	*Bacteria found in the Heart Blood.*	*Bacteria found in the Vein of the Arm.*
1	Bullet wound, chest	4	Streptococcus	o
2	do.	6	Streptoooccus and staphylococcus	o
3	do.	2	——	o
4	Bullet wound, abdomen	4	——	o
5	do.	8	Streptococcus	o
6	Bullet wound, head	10	Proteus vulgaris and staphylococcus	o
7	do.	3	Streptococcus and B. subtilis	o
8	Fracture of frontal bone with cerebral hæmorrhage	7	Streptococcus	o
9	Fracture of base of skull	5	——	o
10	do.	8	Streptococcus, staphylococcus, and B. coli communis	o
11	Disease of valves of heart	3	——	o
12	do.	7½	Staphylococcus	o
13	do.	9	B. coli communis, B. mesentericus, and streptococcus	o
14	do.	6	B. coli communis and B. subtilis	o
15	do.	6	Proteus vulgaris	o
16	do.	10	Streptococcus	o
17	do.	3	——	o
18	do.	2½	Staphylococcus	o
19	do.	8	B. coli communis	o
20	do.	4	Streptococcus	o
21	Rupture of aortic aneurism	6	B. coli communis and staphylococcus	o
22	do.	7	Streptococcus and B. subtilis	o
23	do.	3	——	o
24	Poisoning by oxalic acid	11	Streptococcus	o
25	Pyæmia	8	Staphylococcus pyogenes aureus	Staphylococcus P. aureus
26	Lobar pneumonia	5	Pneumococcus and streptococcus	o
27	do.	6	——	o
28	do.	7	Streptococcus, B. subtilis, and staphylococcus	o
29	do.	4½	Streptococcus	o
30	Peritonitis from perforation of intestine by knife wound	5	Staphylococcus and B. coli communis	o
31	Complicated fracture of cranium	2	Streptococcus	o
32	Cancer of breast and liver	5	B. coli communis and B. subtilis	o
33	Peritonitis following criminal abortion	3	Streptococcus and staphylococcus	Streptococcus
34	do.	7	B. coli communis and staphylococcus	o
35	Traumatic pneumonia following fracture of ribs.	5	——	o
36	do.	8	B. coli communis, pneumococcus, and streptococcus	o
37	Bullet wound in thigh (septicæmia)	4	Streptococcus	Streptococcus
38	Still-born infant	8	Staphylococcus	o
39	Fatty degeneration of heart	6	——	o
40	do.	3	Streptococcus	o
41	do.	9	B. coli communis and proteus vulgaris	o
42	Poisoning by morphine	5	——	o
43	Cerebral meningitis	4½	Streptococcus	o
44	Peritonitis following typhoid ulcer	3	B. coli communis	o
45	Nephritis	10	Staphylococcus and streptococcus	o
46	do.	8	Staphylococcus, B. coli communis, and B. subtilis	o
47	do.	7½	B. pyocyaneus and streptococcus	o
48	do.	9	Streptococcus and sarcina lutea	o
49	do.	3	——	o
50	do.	8	Streptococcus	o

THE SUPPRESSION OF TUBERCULOSIS AMONG DOMESTIC ANIMALS.[1]

By GUSTAV REGNÉR, Ministry of Agriculture, Stockholm.

THE existence of tuberculosis among domestic animals, especially among cattle, is a constant source of anxiety to all who have at heart the interests of a healthy development of animal-breeding as one of the essential conditions of profit in agriculture. It is evident that tuberculosis, when no effort, or at least no serious effort, is made to stop its progress, little by little extends its domain among the cattle. This was shown by Siedamgrotzky, among others, from the statistics of his time; and later statistics do not in general tend to disprove it. The present writer is acquainted with large cattle establishments. which have shown, on a second testing with tuberculin, a considerably greater prevalence of tuberculosis than at the first testing, five to six years before. It has, moreover, been possible to ascertain in Sweden, on different occasions, that the infection of a stock of cattle is accomplished in a comparatively short time.

The close contact of cattle in stalls forms the chief cause of the dissemination of tuberculosis from beast to beast; this was especially insisted upon by Bang and Nocard, and the daily struggle with the pest constantly reveals it afresh.

The denial by R. Koch of the identity between human and animal tuberculosis once seemed likely to destroy an important motive for combat with the latter disease. Researches, however, in later years, in this department of science, seem to have gone convincingly far towards re-establishing the former opinion as to the identity.

Be this, however, as it may, the fact that tuberculosis is the most devastating and damage-inflicting of all plagues among cattle, is more than sufficient to prove the necessity of a suppression of tuberculosis. This, together with the fact that the pest can be suppressed, has long been generally known. There is, on the other hand, some difference of opinion as to whether tuberculosis can be extirpated, and possibly there are still greater differences concerning the method of carrying on the struggle. In the following pages it will be shown what results have hitherto been attained in Sweden in combating the pest; and the conclusion may be drawn from these, that tuberculosis can be extirpated.

In the year 1895 the first steps towards suppression of cattle tuberculosis were taken in Sweden, but a powerful accelerating impulse was first imparted at the end of 1897, when the State took the matter in hand, and granted supplies, which were chiefly employed in free tuberculin tests, and rewards, or, since 1904, in supporting small cattle-owners.

It was soon shown that tuberculosis in Sweden, as in many other countries. had attained a disquieting prevalence among the breeds of cattle, and especially in high-bred strains. In the years 1897-1904, 8698 establishments in all were tested for the first time with tuberculin, with the result, that of the 226, 864 animals of these establishments, 69,717, or 30·7 per cent., proved to be infected with tuberculosis. Remembering the slighter reliability of tuberculin in certain cases it may be advanced with tolerable confidence that about one head in three is tuberculous.

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[1] The remaining abstracts in this number are summaries of papers presented to the VIIIth International Veterinary Congress, Budapest, September 1905.

Considering these statistics more closely, we find that at the first testing 3462 of the stocks above-mentioned, with 46,455 animals, did not respond to the test; the number of tuberculous stocks therefore is 5236, with 180,409 animals, of which 69,717 responded, or 38·6 per cent. tuberculous on an average among the infected stocks. Some of these last do not contain a single animal free from tuberculosis, while others are infected only some tenths per cent.; between these rarely-occurring extremes the frequency of tuberculosis naturally varies a great deal.

Obviously the suppression of tuberculosis has not been undertaken in all these establishments; agriculturists are still far from being sufficiently convinced of, and impressed with, its utility and necessity. It should, on the other hand, be borne in mind that this idea yearly gains ground among the people of the country. The following statistics give an unmistakable proof of the yearly increase in the number of tested stocks. Attention is directed to the fact that in the number are also included stocks from which tuberculosis originally could only be kept away by proper regulations.

Stocks Tested a Second Time.

Year.	*Stocks.*	*Animals.*
1897–98	130	6,970
1899	246	13,151
1900	479	22,113
1901	602	27,016
1902	665	29,451
1903	470	34,473
1904	810	36,149

In the year 1904[1] suppressive measures were undertaken in 810 stocks, with 316,149 animals. These figures are, however, somewhat too low, for a second tuberculin-testing of a stock, or part of a stock, not responding to a first test, is not yearly repeated. In the following I must omit 39 stocks, as their original inoculation statistics are unknown to me.

The result of work continuing, with extremely rare exceptions. two to nine years, has been, that at the end of 1904 we had 34,996 animals, 1646 of which, or 4·7 per cent., responded to the test, as against 33,000 animals with 11,089, or 33·6 per cent., responding, with which the struggle began.

An analysis of these statistical materials yields results of no slight interest in several respects; and it is best if we divide the cattle for this purpose into two groups: partly into those among which it has been attempted to stamp out tuberculosis already existing, and partly into those in the case of which it was a question of exclusion of a tuberculosis-invasion (the defensive in the struggle with tuberculosis).

Among the stock of the first group we meet with such as, at the first testing with tuberculin :—

1. Were tuberculous, but became free from reaction, or
2. Were tuberculous and have since continued to be so. Among the second, such as, at the first tuberculin testing :
3. Were free from reaction, and have continued so, or
4. Were free from reaction, and have become tuberculous.

1. The number of head originally tuberculous, but later free from reaction,

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[1] The statistics for that year are not yet (February 1905) completed, as, possibly, all reports have not been sent in. The number of test-results not yet handed in is in any case so small that it is not necessary to take it into consideration.

is 214 out of 8205 animals, and included at the beginning of the campaign against the pest 8122 animals, with 2652, or 32·7 per cent. reacting. In both cases the average number of head was about 40.

2. In 335 stocks, with 21,202 animals, it has not yet been possible to attain perfect freedom from reaction: 1532 animals reacted, *i.e*, 7·2 per cent.; this, nevertheless, shows a very important success. These stocks contained at the beginning of the campaign 20,040 animals, of which 8437, or 42·2 per cent reacted. It is easy to see that here it was a question chiefly of large establishments; the average number of head in each stock was 63, and far exceeded that of the next category.

3. It was possible to exclude tuberculosis from 170 stocks, or, thanks to tuberculin, to get rid of it where strange animals, or improper-rearing methods for the calves, had introduced it. These stocks contained at the beginning in all 3318 animals; in 1904, 3648; they belong therefore to the class of small establishments. They have been for from two to seven years once annually tested with tuberculin, and among 52 stocks no cases of reaction have appeared at all.

4. The results of the year 1904 conclusively showed that tuberculosis had insinuated itself among 52 stocks originally free from reaction. These, which in the beginning contained 1520 animals free from reaction, but in the year mentioned contained 1941 with 114, or 5·9 per cent. reacting, are in several cases fairly large—an average of about 37 head being therefore shown. The failure indicated by the percentage of reactions mentioned is not of a serious nature, and permits of being easily remedied.

The indirect advantages of this campaign against tuberculosis are, among others, the erection of numerous cowhouses properly constructed and arranged; the increased attention paid to cleanliness of the stalls and treatment of their inmates, the more exact observation of the condition of the animals' health, the yearly disinfection of the stalls, and the greater cleanliness in treatment of milk.

The general impression produced by the adjoined statistics may be stated to be, that considerable success has been attained in the struggle with tuberculosis which has been carried on for many years by hundreds of Swedish agriculturists, and that this result has been achieved by following the methods which Professor Bang in his day laid down as calculated to free the race of cattle from tuberculosis. Naturally, much practical experience has been accumulated in Sweden, throwing a stronger light upon certain points of the method in question, without, nevertheless, inflicting any damage upon its basic principles. This holds true, in the first place, of tuberculin.

Tuberculin is an invaluable and indispensably necessary means for the extirpation of tuberculosis; but he who relies exclusively upon it for differentiating between tuberculous and non-tuberculous animals will sooner or later have bitter cause to regret it. Not only tuberculosis permitting of clinical diagnosis, but also cases where tuberculosis is present in a decided but not very high degree, usually not easily to be detected, may elude the tuberculin test, and in a relatively short time, under favourable circumstances, commit fearful ravages among the stock found free from reaction. Professor Svensson of Stockholm has also proved, by means of a long series of experiments, that tuberculin is somewhat uncertain for repeated tests upon animals once found to react.

These facts render a clinical and bacterioscopic diagnosis imperatively necessary. Suppressive measures must especially be directed against the lung tuberculosis, which is the most important factor in the dissemination in the pest among the cattle-stalls, and in this respect demands much greater attention than the udder variety, which is certainly also of much less frequent occurrence than the former type.

The tuberculin test should therefore not be employed in the case of a stock under grave suspicion of tuberculosis. The proper way to undertake the suppression of tuberculosis among large stocks of cattle is to breed sound stock from the breeding material, in appearance sound, of tuberculous stock. The following rules, supplementary to perfect isolation, infection-destroying treatment of milk food, and disinfection of cowhouses, deserve special emphasis :—

1. That the old stock should from time to time be freed, by the use of clinical and bacterioscopic methods, from individual animals in which tuberculosis has shown itself. 2. That the new born calves of this stock should be immediately brought to the protected stock. 3. That the raw milk should be taken from the mother with such precautions that exterior infection of it should as far as possible be rendered impossible. 4. That the calf should be tested as soon as possible with tuberculin,—the first time, when eight days old, the second time, when half a year old, and afterwards once a year. 5. That every animal so tested and found to react should be marked, to prevent mistakes in isolating, and that a clinical examination should be held upon any animal whose appearance and behaviour (especially in the case of repeated coughing) should give cause for it. 6. That the animals should be exactly numbered (first by ear-marks, later by branding the number on one horn) and entered in books. 7. That animals from other stocks, no matter how young, should only be admitted into the protected establishment from stocks free from reaction, or (in case that this presents unsurmountable difficulties) from other stocks only with certain precautions.

The cattle-trade has a deleterious influence on the suppression of tuberculosis. A single incautious purchase for a stock free from reaction may in quite a short time bring to naught the result of a twelvemonth's, or many twelvemonths', work devoted to rooting out tuberculosis in that stock. The condition generally made, that the purchased animal shall be free from reaction, is not sufficient in itself. Should the purchase come direct from a stock or part of a stock well known to be free from reaction, and which has for years been subject to protective precautions, and should the animal have been born and reared in that stock, then all is well. In any other case, the purchaser must demand such information as to the stock from which the purchase has been made as may clearly show the prevalence of tuberculosis. The stock must therefore be tested, and the result laid before the purchaser. The percentage of tuberculosis among the milch cows, not that among the whole stock, will indicate whether the purchase shall be made or not. It is not practicable to set up a maximum percentage as a guide; the present writer, for his own part, considers that not more than 50 per cent. of the milch stock should be affected. Should the agriculturist decide to purchase, from such a stock, animals free from reaction and which have also been tested clinically, with negative results—and this he should do only with the advice of a veterinary surgeon—he would do well to disinfect the animal after its arrival at his farm, above all with regard to the hoofs and lower part of the legs, and if possible keep it half a year in quarantine. Should local conditions not admit of this, the animal should be placed in a stall, apart from the others, and must not be removed from its place until it has undergone a new testing with a double quantity of tuberculin, which in any case must take place after the interval mentioned. In the matter of oxen, it is naturally very difficult indeed to keep these free from tuberculosis, and therefore the oxen must never be quartered in the cattle-stalls. In this connection it may here be mentioned that the Swedish Government has since 1st November 1903 been occupied in forming breeding - centres for cattle, one of its aims being to promote the trade by means of breeding animals free from tuberculosis.

. . . .

With regard to no infectious animal disease does a greater variety of views prevail, concerning suppressive methods, than with regard to tuberculosis. This is of course principally true of agriculturists, but among veterinarians also there seems to be no agreement as to the utility and necessity of this or that measure. To secure a uniform procedure, lectures for veterinarians on the suppression of tuberculosis would be a good means. Such lectures are given annually in the Veterinary Institute at Stockholm.

As for agriculturists, pamphlets and public notices are of but limited use. The surest means is undoubtedly a talk with the agriculturist in his own house. For, so long as suppression of tuberculosis is a voluntary matter, a powerful propaganda is of the very greatest importance, and the veterinarian is the best man for this purpose. But the state should also undertake this propaganda, and that by means of free lectures, as has been done in Sweden, an adviser having been instituted for the purpose.

This Tuberculosis-Consultant (hitherto Veterinary Surgeon O. Stenström has held this position) has the principal duty of assisting cattle-owners, without any charge whatever, with advice and instructions, at request, on the spot, regarding the extirpation of tuberculosis from the stalls, and in concert with the local veterinary surgeon.

The struggle against cattle tuberculosis is in Sweden a voluntary effort of the agriculturists under the guidance and with the co-operation of the state, with the aim of extirpating the pest.[1] That this object is attainable, appears to the present writer to be beyond all doubt, even though a pest for many decades allowed to spread unnoticed among the cattle of a country naturally needs many years of hard and continuous work before it is brought to extinction.

Should the campaign against cattle tuberculosis be solely directed to keeping the plague within certain limits, to a qualitative restraint in fact, as seems to be the case in different Continental countries, then naturally a portion merely of the rules laid down in Bang's programme will be found sufficient. Every one of these, and also every combination of them, produces, if consistently and perseveringly carried out, a diminution of tuberculosis; and in this respect the process which Ostertag recommends against this pest will certainly bear fruit among the stock of cattle belonging to the East-Prussian-Dutch Herd-book Company. Only, stock free from tuberculosis are not to be obtained either through this or any other combination of the above-mentioned rules.

But to what does all this tend? We have even come so far that we can seriously discuss, at an International Veterinary Congress, the question of immunisation of cattle from tuberculosis. The decisive word has been spoken in the struggle against this pest. As, however, this question will be especially treated at the Congress, the present writer confines himself to expressing the certain conviction, that what he has here advised will not in the main be found superfluous.

The author regards the following points as being of fundamental importance:—

1. Notwithstanding that tuberculin diagnosis is of invaluable use for the suppression of cattle tuberculosis, it must nevertheless, when employed with a negative result, be supplemented, in all cases where the slightest reason therefor exists (chronic cough, udder maladies, leanness, barrenness, etc.—purchased animals), with clinical and bacterioscopic diagnosis. The State must supply the necessary means for this.

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[1] The Swedish Parliament has hitherto (incl. 1905) granted in all 485,000 Kronen for prosecuting the struggle (a Krone = 1 Mark 12 Pfennig). This sum does not include the cost of suppressive measures ordained by a special Act against udder tuberculosis, which cost was defrayed out of other moneys of the State.

2. Lung tuberculosis is, of all infectious forms of tuberculosis, the principal spreader of the pest among the stalls, and must therefore be combated according to the principles legally established in Sweden and Denmark for udder tuberculosis, and also by means of compulsory notice (or, still better perhaps, compulsory examination of cattle), compulsory slaughter, and compensation to cattle-owners from the funds of the State.

THE METHOD OF INFECTION IN TUBERCULOSIS OF THE DOMESTIC ANIMALS.

By Chief Veterinary Surgeon BONGERT, Superintendent of the Bacteriological Laboratory of the Municipal Abattoir at Berlin.

TUBERCULOSIS has long been held to be a hereditary disease. Two possibilities of the hereditary transmission of tuberculosis are conceivable: the generative or conceptional, and the placentary transmission. No really generative transmission of tuberculosis either from the father's or the mother's side has hitherto been established, either experimentally or from observations not open to objection. On the other hand, the placentary transmission of tuberculosis to the fœtus has been observed in a great number of cases among cattle and human beings, and has also been experimentally produced by several investigators. Characteristic of placentary infection with tuberculosis is the localisation of the tuberculosis in the liver and the portal glands, in which the most extensive and the oldest disease-centres are to be indicated. This form of tuberculosis, which gives a complete impression of primary origin, is observed not at all infrequently among older calves and young cattle, and is to be regarded as congenital tuberculosis (Bang, Thieme). Only in far advanced forms of tuberculosis can transmission from the mother to the fœtus occur, and that according to Nocard and Bang, in about one per cent. of the cases. Hereditary transmission is not therefore to be regarded as an etiologically important cause of the genesis or propagation of tuberculosis. This holds good also for the hereditary tendency. Acquired tendency has a much greater importance in the genesis of tuberculosis, for through this (lesions of the skin and especially of the mucous membrane) the entrance of tubercle bacilli into the body is facilitated, and in many cases first rendered possible. The most important agent in the genesis of tuberculosis is post-natal infection. Those predisposing causes which arise from unfavourable economical conditions and are adapted to lower the resisting power of the body (among these, long confinement in houses, over-production of milk, etc.), are only so far regarded by the present writer as important, partly as they provide occasions for the admission of tubercle bacilli (position in the byre), partly as they unfavourably influence the course of a tuberculous infection already existing.

With calves and pigs, intestinal infection, from the administration of milk containing tubercle bacilli, plays the chief part; with grown cattle, ærogenous infection. Von Behring's theory of the genesis of tuberculosis, making infantile intestinal infection the chief cause of lung tuberculosis which appears later, has not the importance which von Behring attaches to it.

Of an inferior importance in the transmission of tuberculosis is infection from the outer skin, the genital apparatus, and the udder.

Lung tuberculosis also develops secondarily, as a rule, by reception of tubercle bacilli from the mucous membrane of the respiratory organs (ærogenous infection), for these bacilli pass through the mucous membrane without leaving lesions at the points of entry. The first disease-centres are in the bronchial glands, and thence affect the lungs through the circulation.

The writer draws the following conclusions:—

1. No generative inheritance of tuberculosis from the father's or the mother's side has hitherto been proved, either by experiment or by unexceptionable observations; placentary inheritance, on the other hand, has been firmly established.

2. Tuberculosis is inherited by descendants only in a slight percentage of cases; in by far the greater number of cases tuberculosis is of extra-uterine origin.

3. Inborn and acquired dispositions play only an unimportant part in the genesis of tuberculosis among domestic animals. Unfavourable economical conditions, calculated to lower the resisting power of the body, are less adapted to facilitate infection than unfavourably to influence the course of already existing latent disease. Infection, *i.e.* the penetration of the tubercle bacillus into the tissues of the body, is to be regarded as the most important agent in the genesis of tuberculosis.

4. As points of entry for the tuberculous infection, the mucous membrane of the organs of respiration, and that of the intestines, come chiefly into observation. The transmission of tuberculosis by means of coition or through infection of a wound of the skin, or through the openings of the udder, is of slight importance.

5. Intestinal infection chiefly occurs among calves and swine, as these animals are, above all, liable to take up tubercle bacilli with their food. This mode of infection is also the rule among flesh eaters and poultry, less frequently among horses.[1]

6. With older cattle, the transmission of tuberculosis mostly takes place through ærogenous infection, in consequence of sound animals living in close contact with diseased ones in byres. The prevalence of tuberculosis of the bronchial glands and the lungs, among older cattle, indicates this.

Tuberculosis of the lungs develops itself in most cases by means of the circulation of the blood, from the primarily diseased bronchial glands.

PROTECTIVE INOCULATION AGAINST TUBERCULOSIS OF CATTLE.

By Professor Dr HUTYRA, of Budapest.

OF all the attempts hitherto made to render cattle artificially immune to tuberculosis, the protective inoculations of von Behring unquestionably merit adequate consideration in the first instance; for they are founded upon scientifically based theoretical conceptions, and have, moreover, already been applied in practice to more than 10,000 animals.

Experience has proved that no difficulties are encountered in carrying the process into practice, and that it is entirely innocuous to animals previously sound. It has also been shown by control experiments that this protective inoculation very materially increases the power of the cattle to resist subsequent artificial infection with bovine bacilli. In especial, the experiments of the present writer have demonstrated that cattle, treated beforehand in exact accordance with von Behring's method, undergo without injury intravenous injections of virulent tubercle bacilli, in such quantities as would be absolutely fatal to animals not inoculated; and further, that the immunity

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[1 "This is a curious error, as intestinal infection is the almost exclusive method of infection in equine tuberculosis." Eds.]

conferred by the inoculation continues unimpaired for eight months after the process.

The question, however, whether the increased power of resistance thus conferred holds in the case of natural infection does not as yet, from the hitherto available results of experiments, admit of final solution. The tuberculin tests applied with a view to solving this problem gave results which failed to satisfy the expectations cherished *a priori.* That is to say, that in several places a considerable percentage of the previously protectively inoculated cattle reacted, between which percentage and that proportion of the non-inoculated cattle which reacted no appreciable difference appeared.

Whence arises the contradiction between the considerable power of resistance of protectively inoculated animals to artificial bovine infection, and the not very favourable results of the tuberculin tests. What the practical value of the von Behring protective inoculation may be is a question to which only continuous, accurate, and practical observations can furnish the desired answer.

The power of resistance of cattle to artificial bovine infection is also considerably increased, as the experimental researches of von Baumgarten and the present writer have proved, by a single subcutaneous injection of human tubercle bacilli. Should these observations be confirmed by further ones, this method, owing to its simplicity and easy applicability, would deserve further testing. Up to the present, observations have been wanting with regard to the degree of resistance offered by cattle so treated to subsequent natural infection.

Conclusions.—An intravenous injection, once repeated, of human tubercle bacilli, after von Behring's method or some similar one, increases to a very considerable extent the power of resistance of cattle to artificial infection with bovine bacilli.

The process is innocuous to sound cattle, and presents no difficulties in carrying it into practice.

The question whether, and if so, how far, the immunity produced in this way extends to natural infection, finds no solution in the results of past experience; to solve this problem accurate observations of inoculated animals, continued for years, will still be necessary.

A similar inoculative protection against artificial infection is apparently afforded by a single subcutaneous injection with cultures of human tubercle bacilli.

THE IMPORTANCE OF THE ACID-PROOF BACTERIA IN THE DIAGNOSIS OF TUBERCULOSIS.

By Dr A. Aujeszky, Docent at the Veterinary College at Budapest.

Since the discovery of acid-proof bacteria resembling the bacillus tuberculosis, the search for the tubercle bacillus has become a more difficult task than before, when all bacteria found sufficiently acid-proof were, on microscopic examination, considered to be tubercle bacilli. But between these latter and the acid-proof bacteria which resemble them, there exist essential differences as well as similar attributes, and these differences allow of the differentiation, now with greater, now with less facility, between the tubercle bacilli and the others.

By their morphological characteristics only, those acid-proof bacteria whose microscopic images show marked differences from tubercle bacilli may be distinguished; that is, the very short, thick, cocco-bacilliform, acid-proof bacteria, as for instance the acid-proof bacillus of Preisz.

There is no *colour-reaction* known by which certain acid-proof bacteria can with absolute certainty be differentiated from the tubercle bacillus; for there are acid-proof bacteria which offer to acids and alcohol as high a degree of resistance as the tubercle bacillus. In such cases, as the author has convinced himself in comparative researches, many colouring methods (such as those of Grethe, Marzinowski, Weichselbaum, Honsell, Bunge, and Trautenroth) give us no aid, although these methods may otherwise be of great use in distinguishing many acid-proof bacteria. The degree of resistance to acids may also vary with one and the same acid-proof bacterium under different conditions; the quality of the fostering soil, the age of the culture, as well as the circumstance whether the bacterium has been in an animal organism or not—all these influence the degree of resistance to acids. Thus the author can confirm the statement of Möller regarding the "*Grass-bacillus*" and that of Karlinski regarding the bacillus described by that investigator—that the bacteria of a youthful stage are more acid-proof than later. Likewise he has found, as have others, that the resistance to acids of many bacteria (Karlinski's bacillus *Streptothrix farcinica*, Markl's *Streptothrix* from butter from the Vienna market) increases in animal organisms, while that of others on the contrary decreases. (Thus, for instance, Möller's *Grass-bacillus* as also the milk fed acid-proof bacillus and the smegma bacillus of the same investigator, and Korn's *Bacillus friburgensis*, have lost more or less of their resistance to acids when in the organism of the frog; in some cases he observed that *Bacillus friburgensis* taken from the blood of the grey mouse and cultivated on agar, as well as Möller's "Pearl-disease bacillus" and Marpmann's bacillus, reared in the abdominal cavity of the guinea-pig, became far less acid-proof; which difference, however, disappeared in the second generation reared on agar).

A great portion of the tubercle-like, acid-proof bacteria are distinguishable from these by their peculiarity of growth, principally by their rapid increase of size, their slighter requirements as to temperature and fostering soil, as well as by their production of colour; *in general, however, it is a question of gradual differentiation only.* There are acid-proof bacteria which develop just as slowly, and demand as high a temperature with regard to mammals, as the tubercle bacilli of mammals. And, on the other hand, we know that the tubercle bacilli can increase rapidly on certain media, and it is certain that they may be accustomed to grow at a low temperature. On the other hand, the author has found that the bacillus of fish tuberculosis gradually allows itself to become accustomed to the body temperature of mammals, and then recovers the attributes of the mammal tuberculosis bacillus.

Also by their perceptible *pathological effect* on animals experimented upon essential differences are shown, in general, between the tubercle bacilli and the acid-proof bacteria which resemble them; but the similarity in the appearances of disease requires the greatest care in judging the respective cases.

On the whole, it may be said that several acid-proof tubercle-like bacteria resemble the tubercle bacilli in such measure that they may be distinguished from them only by exact comparison of *all characteristics*; most, however, are comparatively easy to differentiate from the tubercle bacilli.

The numerous common characteristics of the tubercle bacilli and the acid-proof bacteria which resemble them point to a *near relationship* of all these micro-organisms; but at the present stage of knowledge it can only be said that the tubercle bacilli and the acid-proof bacteria which resemble them form a kindred group. But, on the other hand, we are not at present justified in claiming the latter as tubercle bacilli in the saprophytic stage.

The author observes, in conclusion, that it would appear to be best, in the interests of simplicity and the avoidance of mistakes, to designate the acid-proof bacteria which resemble the tubercle bacillus as "Paratuberculous bacilli."

ARE THE TUBERCLE BACILLI OF MAN, MAMMALS, AND BIRDS DIFFERENT IN KIND OR NOT?

By Professor Dr H. PREISZ, Budapest.

THE experiments which have been made for the purpose of comparing the characteristics of tuberculosis in human organisms and in cows have demonstrated that the tubercle bacillus of the cow is indeed distinguished for the most part by a higher grade of virulence as well as by some insignificant morphological and cultural characteristics; but they have also demonstrated that there are also tubercle bacilli of human origin which can in no respect be distinguished from those of the cow.

More important differences have been established between the tubercle bacilli of mammals and the typical tubercle bacilli of birds, as the latter class of bacilli form moist mucilaginous cultures, in the case of most mammals do not produce tuberculosis, and possess a higher temperature optimum. Bacilli of the character of the tubercle bacilli of mammals have, however, been found in birds; and, *vice versa*, in birds have been found bacilli of the character of the tubercle bacilli of mammals.

If the differences between the various classes of tubercle bacilli were constant, the assumption that they belong to different species would not be unjustifiable, and we should explain, for instance, the occurrence of tubercle bacilli of the bovine type in men or in birds simply by assuming that men and birds were alike susceptible to the bovine virus. The differences, however, are not constant, for there are all kinds of transition forms among the typical tubercle bacilli of man, mammals, and birds.

The degree of virulence of the tubercle bacillus of man is not always the same as that of the tubercle bacillus of the cow, but varies within a wide range. In general the tubercle bacillus of mammals is not dangerous for birds; and still, in exceptional cases, bacilli of this character have been found in birds. The virulence of the tubercle bacilli of birds has also proved to be not uniform. Moist mucilaginous cultures, such as the tubercle bacilli of birds often yield, are also no constant characteristic; in exceptional cases the cultures of the tubercle bacilli of birds are similar to those of the tubercle bacilli of mammals, without, however, approaching them in respect of virulence.

The virulence of the tubercle bacillus has been remarked to increase as a consequence of its passage through the animal, and, on the other hand, the virulence has been seen to diminish when the bacillus has been long developed by means of artificial nourishment. By employing suitable methods cultures of the tubercle bacillus of the mammal can acquire the characteristics of that of the tubercle bacillus of the bird, and *vice versa*.

In the cultures of all tubercle bacilli tuberculin is produced; moreover, tubercle bacilli of different origins stand in closest relationship with regard to their reciprocal agglutinating properties and power of establishing immunity.

According to all this, tubercle bacilli in general are to be regarded as changing micro-organisms, and, accordingly, the tubercle bacilli of men and birds, as well as those of cattle and other domestic animals, are to be regarded as variations of a single species of bacteria, which, in the course of time, have become differentiated by passing into different animals. The variations which often occur among the types of the individual varieties are examples of germs which have not had time and opportunity to become transformed into the typical varieties.

Hence it follows that the tubercle bacilli of any one species of animal cannot be regarded as not dangerous for other species or for mankind, and that a rational treatment of the disease, to be successful, must not be limited to one species of animal, but must extend to all classes of animals which are affected by tuberculosis.

MILK AND DAIRY PRODUCTS AS PROPAGATORS OF TUBERCULOSIS.

By Dr Aladar de Fáy, Inspector in the Health Department of the Royal Hungarian Ministry of the Interior, Budapest.

The answer to the question whether milk containing tubercle bacilli of animal origin is capable of producing tuberculosis of a typical character in the human organism, will be rendered possible by a satisfactory determination of the following questions of detail, which are all intimately connected with the main question.

With regard to the human organism which receives the infecting matter, it is first of all necessary to determine whether bacilli of bovine origin can infect the organism if, by the agency of nourishment, they are introduced into the intestinal canal.

As it is not possible to experiment on the human frame to determine this point, we are compelled to seek proofs from statistical data on the subject, concrete empirical facts which have been placed on record, and pathological changes observed in the human body.

In judging the nature of the question, no satisfactory standpoint is afforded us by the statistical data, nor do these data supply us with proofs that the prevalence of tuberculosis in cattle or the quantity of milk consumed, stands in causal connection with the prevalence of tuberculosis among human beings. Moreover, the statistics relating to intestinal tuberculosis are of no particular value, as, on the one hand, the infection may be produced by bacilli of human origin, a circumstance which it is often impossible to perceive, and, on the other hand, the accounts of the different authors are so divergent as to the occurrence of the disease that it is at present impossible to obtain a clear and satisfactory view with regard to the prevalence of the disease.

The concrete cases placed on record and adduced as examples of infection are likewise of only secondary value as proofs, for in the case of infections found in nature the conditions may be extremely varied and complicated. The quantity and the virulence of the infecting matter, the various methods of treating and using the milk, the quantity of milk consumed, the frequent recurrence of the opportunity of receiving infection, etc.,—all these are factors which, co-operating with the resistance-capacity of the individual organisms, lead to the most divergent results.

As no exact pathological data or results of experimental researches are at our disposal, accounts of the kind are, by reason of their defectiveness, unsuited to the solution of the question, and prove generally the human origin of the infection just as little as they are able to demonstrate its bovine origin.

On the other hand, we may regard as decisive proofs those pathological discoveries and the results of experiments in this connection made on cattle in the Imperial Health Department at Berlin by Kossel, Weber, and Henn.

It is well known that Koch has demanded, as a criterion of the animal origin of tuberculosis observed in man, proof of the fact that an ox which has been subjected to a subcutaneous injection of virus obtained from a human being becomes the subject of universal tuberculosis; for, in his opinion, only bacilli of bovine origin produce disease in such a manner, whereas the bacilli of human origin are capable of producing in cattle only a local and curable tuberculosis.

The above-mentioned authorities have actually succeeded in obtaining this result with six out of fifty-six cultures of tubercle bacilli of human origin—that is, in more than 10 per cent. of the cases. The bacilli, with the exception of a single group, were all derived from the dead bodies of children under seven years of age, being taken from tuberculous ulcers in the intestines, the mesenteric glands, or from the lungs.

The above-mentioned authorities have found that the differences existing in the morphological and cultural qualities of bacilli of bovine origin and those of human origin are, though small in number, so constant in character that they consider there is good ground to assume the existence of distinct types of bacilli, human and bovine. In the above-mentioned cases of human tuberculosis the presence of bacilli of bovine origin was also demonstrated. Hence it may be regarded as an established fact that food containing bacilli of bovine origin produces tuberculosis in the human body.

With regard to man, there accordingly arises the further question, as to whether a tuberculous infection of the human organism can also be produced through the intestinal canal in the case where the walls of the canal are normally constructed and undamaged, or only in the case of certain changes brought about by illness.

Experiments relative to the behaviour of bacilli during the process of digestion, carried out by Porcher and Desonbury, and in recent times by K. Rogosinsky, J. Barthel, Ostertag and Nebelthau, and by Nicolas and Descos, as well as experiments made in feeding apes, the animals of closest affinity to mankind, by Grünbaum, Nocard, Cipollino, De Haan, and MacFadyean relative to the transmission of tuberculosis, provide us with a decisive answer to the above question. According to these experiments, the bacilli of tuberculosis can penetrate the undamaged walls of the intestines without infecting the walls, and can produce the first symptoms of tuberculosis in organs which lie far removed from the place where the infecting matter first entered the body, generally in the so-called predilection organs, which are the most sensitive to tuberculosis. No doubt can exist that tubercle bacilli are capable of penetrating the walls of the intestines of the human body, just as was the case with the apes which received milk infected with tubercle bacilli. This fact has, moreover, been fully confirmed by pathological discoveries described by Kossel and his companions, according to which bacilli derived from tubercular mesenteric glands, and in one case from a lung, have proved to be of bovine origin, although in these cases no trace of tuberculosis had appeared in the structure of the intestine.

In the case of sucklings this penetration of the intestines by the bacilli, and consequently the possibility of remote organs being infected, is facilitated through the circumstance that, in the intestines of sucklings, the connecting epithelial layer and the protecting mucous membrane are still undeveloped; but these, as well as the ferment-secreting glands in adults, are only seemingly preventive factors, and not capable of excluding infection with any degree of certainty.

The assumption that tubercle bacilli of bovine origin, taken with food and transmitted through the walls of the intestinal canal, can produce tuberculosis in the lung, the organ of man predisposed for the infection, is to all appearances proved by the above mentioned observations of Kossel, Weber, and

Henn, who derived tubercle bacilli of bovine origin from the lung of a five-years-old child in whose intestines no trace of tuberculosis was perceptible.

In order to obtain a clear conception as to tuberculous infection arising through the agency of the milk of animals, it is necessary, moreover, to determine in what relation the locality and degree of the tuberculous infection stand to the secretion of the tubercle bacilli resulting from the milk. There is no doubt that the milk containing the most bacilli, and consequently the most dangerous milk, is derived from animals which suffer from tuberculosis of the udder; but, according to the results of the more recent experiments of Rabinowitsch, Kempner, Ravenel, MacWeeney, Moussu, Gehrmann, Evans, and Mohler, we must also regard with mistrust the milk of cows which not only do not suffer from udder-tuberculosis, but disclose no illness when clinically inspected, the hidden tuberculosis of the animal being alone manifested by the reaction following tuberculin inoculation.

According to the experiments of De Michele, Michelazzi, Jemma, and Ripper, the milk of cows infected with tuberculosis is to be regarded as injurious on account of the toxin it contains, even in the case where it contains no living tubercle bacilli.

Numerous experiments have proved that tubercle bacilli in an active condition occur in the milk, butter, butter-milk, margarine, and other products of milk in general consumption. Fischer, Galtier, Heim, Gasparini, Lafar, Laser, Dawson, Hamilton, and others have also demonstrated that the tubercle bacilli in the various milk-products retain their virulence for a considerable length of time, and consequently such products, if prepared from the milk of a cow infected with tuberculosis, are to be regarded with suspicion.

As tubercle bacilli of bovine origin are proved to occur in the human organism; as in the intestinal canal of human beings, especially in the case of sucklings, the conditions enabling the bacilli to penetrate the walls of the canal are always present; and as the milk of cows infected with tuberculosis can also contain virulent tubercle bacilli even when the udder of the animal is in a healthy state, it follows that the milk of cows suffering from tuberculosis must be regarded as undoubtedly capable of producing infection. But as to how frequently such cases of infection occur in practice, no definite conclusion can be drawn at present, owing to the fact that the data are in part defective and in part contradictory.

MILK AND ITS TREATMENT, WITH ESPECIAL REGARD TO THE REFORM OF MILKING IN ACCORDANCE WITH HYGIENIC REQUIREMENTS.

By Dr Gruber, Kiel.

From the application of centrifugal force, and the introduction of the centrifuge, the industry of dairy farming received a powerful impulse towards rational methods. For milk and its products, applied chemistry followed closely upon physics; the methods of determining milk fat developed and continued to be ever more generally applied, so that to-day the method best adapted for practical use, that of acidbutyrometry, has become the common possession of the whole industry, while the refractometric method of Wollny has been more especially adopted in the testing departments.

The frequent occurrence of rapid "turning" of milk has directed the researches of many observers towards the origin of this process; and, now that it has been ascertained to be due to the activity of micro-organisms, the value of bacteriology to the milk industry has been fully recognised. Gradually increasing knowledge of the several effects of different groups of bacteria upon milk has brought into prominence the following questions: Where are the sources of milk-infection to be sought for? How are the physiological influences of the bacteria to be checked—or, in other words, how can milk, which so readily spoils, be rendered proof to injurious influences? Besides the micro-organisms of a harmful character, there must be others possessing useful properties; and, indeed, the bacteria which produce lactic acid, having proved to be of this kind, now play an important part in the production of butter.

The reply to the two questions stated above was, for the collection and further treatment of milk, conclusive from a hygienic point of view. Cleanliness in every respect and every manipulation of the milk, till it reaches the consumer, is the first and most important principal for obtaining milk which will keep. Cleanliness on the part of the staff of the dairy in their clothing, which should be adapted to the work, and a perfectly sound constitution on the part of every milker are points not to be overlooked. Before milking, the udder and adjacent parts of the belly should be rubbed with a damp linen cloth, and all necessary utensils, perfectly clean, having been put in their proper places, the hands of the milker must be carefully washed, before the milking of every cow, with lukewarm water and soap. Milking must be done with dry hands only; wetness of the hands must, owing to facility of infection, be absolutely avoided. The first three or four jets should be thrown out. The milk, when obtained, must at once be removed from the dangerous atmosphere of the cowhouse, brought into an airy room, and strained to free it of foreign bodies. The feeding of the cows at milking time must in any case be prohibited, the fodder being rich in micro-organisms capable of destroying the keeping qualities of the milk; and the carrying out of dung at that time is equally unallowable.

The stabling should be so arranged that fresh air can always enter and polluted air go out, that sufficient light is always to be had, and that the foundation of the cowhouse is not exposed to the penetrating wet. Care must further be taken to remove animal excrements from the air of the cowhouse as soon as possible The stall for each cow must be sufficiently large, and the animals must always have enough pure water as they require it. Cleanliness and care in every point connected with the stabling are the chief necessities for obtaining milk which will keep well and be hygienically unexceptionable.

In the dairy the milk should be cooled as much as possible, either with water in a cooler or by means of machines; the cooler the milk is when put into the transport cans, the cooler it reaches the consumer, and the greater the guarantee for its keeping well. To preserve the milk, heat may be employed as well as cold; it may be pasteurised at 80-85° C., or sterilised at 98° C., perhaps at 100° C. or upwards. Both the latter methods produce more or less change in the raw milk; the albumins coagulate, the dissolved calcium salts are precipitated, the milk sugar is caramelised, the milk acquires a disagreeable taste, and its utility for cheese-making purposes is much decreased, while its marketable quality is lost. Perfectly sterile milk can only be procured under pressure, at a temperature of over 100° C., and is chiefly valuable for scientific investigations.

SUPPRESSION AND EXTIRPATION OF RABIES.

By Professor M. CASPER, M.D., Breslau.

THE question of checking rabies has not come into discussion since the second International Veterinary Congress at Vienna in 1863. At that Congress, the committee resolved "*That the introduction and strict execution of a rational supervision of dogs was the surest means* of preventing and ultimately extirpating rabies, and that, consequently, the institution of such a supervision, as far as possible uniform, in the different countries was to be disired."

The plan proposed by the present writer for such a supervision of dogs was accepted by the Congress in the form returned in the chief report.

The question of suppressing rabies has not come into discussion at any subsequent Congress. This may be ascribed to the number of cases of rabies having markedly decreased till about the middle of the nintieth year (1994), and to the opinion prevailing, that the disease could be limited, and finally extirpated, by means of the existing legal regulations. But *from the middle of the ninetieth year* there was, in almost all countries, *a grave increase* in the numbers to record; so that at present (except in Great Britain and Switzerland), rabies prevails to a disquieting extent.

The present writer gives, from carefully compiled tables, a view of the numerical distribution of lyssa in the different countries, and of the prevalence of rabies cases among animals within the last decades. The most exact figures relating to all sorts of domestic animals are those returned from the German Empire and Hungary. In Prussia, as the statistics show, *the Eastern Provinces, especially the districts on the Austrian and Russian frontiers*, have long been the most infested with hydrophobia; furthermore, those districts of the Kingdoms of Saxony and Bavaria which lie on the frontier of Austria are also much infested.

According to statistics, *France*, *Hungary*, and *Germany* are next to be regarded as much infected. Hydrophobia is exceedingly prevalent in Russia, but exact statistics for that country are not to be had. Austria, especially Bohemia and Galacia, Belgium, Italy, Spain, and Roumania have also numerous cases of rabies to report.

In Great Britain of late years the number of animals affected by rabies has been continually decreasing; this is to be attributed to stringent regulations and a special law, forbidding the import of dogs except under six months' quarantine.

In the suppression of rabies, the tasks incumbent on the veterinary police are, to lessen as much as possible the chance of infection among men and animals, thus limiting the dissemination of hydrophobia; and to destroy the sources of contagion. The dog is notoriously the chief disseminator of rabies; the danger of infection with this disease would therefore be most surely removed if it were possible to send every dog out of the world. As this is not practicable, and as the proposal made by some, to submit all dogs to obligatory protective inoculation, is also, for reasons fully gone into by the present writer, impossible to carry out in practice, we must content ourselves with those measures which tend, firstly, to *decrease the number of dogs* as much as possible, and, secondly, to limit their *uncontrolled roamings* and their *opportunities for biting*. To attain the former object, we have the dog-tax in connection with a control of dogs, while biting and roaming about at will may be limited respectively by the *muzzling order* and *confinement*, or *leading with a string*. In addition to these general regulations, special provisions should be put in force whenever required by the presence of the epidemic, as provided in the *laws relating to Infectious Diseases*.

The Dog-Tax.—The dog-tax has been generally introduced into several countries, while in others its adoption is a matter for local authorities. The imposition of a *general dog tax*, *sufficiently high*, produces an effectual diminution in the number of dogs. But it is only effectual when sufficiently high and connected with official control of dogs, so that evasions may be punished. Experience has shown in many countries that the dog-tax there was too low, and had only a passing effect in decreasing the number of dogs (England, France, Prussia, Saxony, Baden). The best effects of the dog-tax are to be recorded for Bavaria, as the present writer shows in detail.

The Muzzling-Order.—The muzzling-order is an important and excellent means of suppressing rabies; when properly drawn up, it prevents biting, and therefore the possibility of infection of men and animals. The objections urged against the muzzle are unjustifiable. As with the dog-tax, so success attends the muzzling-order only when the regulations are strictly carried out; when all dogs encountered without a muzzle are impounded, and either confined or destroyed.

Obligatory muzzling, on the appearance of epidemic hydrophobia, has everywhere been always attended with favourable results; as is shown fully by examples from Berlin. Wurtemberg, and Saxony.

Legislative Regulations at Periods of Prevalence of Hydrophobia.—Most civilised states possess laws for the suppression of animal epidemics, which laws coincide in the essential points. The chief points of the regulations for the suppression of hydrophobia, current in the German Empire, are the following:—

1. *Obligatory Notification* by owners in case of hydrophobia or suspicion thereof.
2. Measures before the action of the police; confining or killing of the rabid or suspected dog or cat
3. Police ascertainment of the fact of an outbreak.
4. *Keeping dogs in confinement.*
5. Compensation for domestic animals destroyed by order of the police (but not for dogs).
6. Institution of penalties.

The success of the law relating to infectious diseases varies in the different countries. In *Germany* the legal provisions have on the whole proved adequate and effective; this is illustrated by several facts. Only in the border districts do cases of hydrophobia, due to the entrance of rabid animals from neighbouring countries, continually recur.

The results of legislative enactments would be decidedly more favourable still, if the regulations were carried out with sufficient strictness. The fault of this lies partly with the public, partly also with the executive.

That hydrophobia can be successfully combated by energetically carrying out the police regulations, while on the contrary their neglectful execution entails the wider spreading of the epidemic, is most obviously shown by the history of hydrophobia in England and in Paris (here the writer goes into full particulars).

The question now arises, what regulations to adopt against hydrophobia from the point of view of international suppressive measures against epidemics, so as to limit and finally to extirpate it; and whether, and if so how far, existing enactments need improving.

As the *first requirement* it can hardly be sufficiently emphasised that *suppression in every country* should be based on the same principles, and carried out with the same strictness. Particularly against rabies is no Continental country capable of protecting itself, since it is impossible to keep it out at the frontier.

The duty of compulsory notification, hitherto prescribed only for cases of rabid or suspected dogs, must be extended to include cases of animals bitten by such dogs. Not only the owners, but all persons having any knowledge of the case, must be obliged to notify.

Finally, it is to be considered whether the legal limitation of the cordon to 4 km., and of the duration of quarantine to three months, is not too narrow.

The writer, on the grounds he has explained, proposes the following final resolutions:—

I. Measures for the suppression and final extirpation of rabies in a

Continental country can only be successful when the veterinary police protective regulations in neighbouring countries are also adequate and strictly executed. It is therefore urgently necessary that the action of the veterinary police in all countries, with regard to hydrophobia, should be based on the same principles.

II. Compulsory notification, hitherto held necessary only for cases of actually rabid or suspected animals, must henceforth be extended to animals bitten by rabid or suspected dogs. This obligation shall be incumbent, not only on the owners, and on all persons included under Section 9 of the law of Infectious Diseases, but also on all such persons as know that animals have been bitten by such dogs.

III. It is to be considered whether it be not desirable to extend the quarantine for dogs to a wider space than hitherto, and to a longer time than three months.

IV. It is desirable that a law, equally valid in all countries, and strictly executed, should be introduced, for the control of dogs; and that this law should contain the following provisions:—

1. Every dog in town or country, without exception, is to be registered, and entered in a list for assessment.
2. All dogs registered are to be provided with a badge on the collar, such badge bearing the name of the owner and the dog's number in the assessment list.
3. All dogs are to be provided with a well-fitting muzzle, so constructed as to render biting impossible, but not preventing them from eating and drinking.
4. Dogs without badge or muzzle are to be impounded, and, unless claimed within a certain period, destroyed.

RULES FOR JUDGING THE REACTION TO MALLEIN.

By M. J. Tátray, Veterinary Inspector, Budapest.

In the task of accomplishing a total extirpation of glanders in infected studs, and that in the shortest time and at the smallest material sacrifice, mallein, as a means of diagnosis, plays an important part.

It has been found by practical experience that concealed glanders may develop into the pronounced form in an infected horse within six months following the infection.

This circumstance, as well as the necessity of establishing a diagnosis for animals suspected of suffering from glanders, rendered necessary, in Hungary, the ministerial ordinance, whereby the mallein test must be made in the infected studs by employés of the State.

This decree provided, according to the circular ordinance of the Minister of Agriculture in 1900, No. 4000, that suspected animals, in case the mallein reaction were found to occur, were at once to be destroyed; while, in case it did not occur, they escaped quarantine, with this limitation, however, that they might not for sixty days be sold into any other district. The animals reacting uncertainly must, in extended quarantine, be treated with mallein a second time after sixty days, and eventually a third time after a further sixty. At present, however, a third mallein test is never undertaken, as animals giving the typical reaction are destroyed—seldom after the first mallein test merely, but in any case after the second, while the uncertain or atypically reacting are released, under the restriction mentioned above, from quarantine.

The mallein test for animals suspected of glanders is always obligatory; on the other hand, those suspected of infection are treated with mallein only with the consent of their owners. But, save in some exceptional cases, owners usually make no difficulty about handing over animals liable to infection.

In Hungary during 1901, 1902, and 1903, 458 horses suspected of glanders, and 2818 suspected of infection, underwent the mallein test.

Of those suspected of the disease, 379 were destroyed in consequence of the results of the test; the *post-mortem* examination proved that 366 (93·9 per cent.) of them were glandered.

Of the 2818 horses suspected of infection, 1849, *i.e.*, 65·6 per cent., which were found at the first test absolutely non-reacting under mallein, were released from quarantine. At the second mallein reaction, 550 (or 70·8 per cent.) of the infected stock of 776 head were released from quarantine, on account of total absence of mallein reaction, or uncertain and atypical reaction respectively. At the third mallein test 31 head (43·6 per cent.) of an infected stock of 71 were released, likewise owing to uncertain and atypical mallein reaction, or to total absence of any, respectively.

After a thrice-repeated testing with mallein, 2441 head (86·7 per cent.) of the infected stock of 2818 were freed from quarantine.

On the other hand, 377 head (13·3 per cent.) in all, of the infected stock of 2818, were destroyed at the first, second, and third mallein tests. 344 head (91·2 per cent.) of these 377 were found, *post-mortem*, to be suffering from glanders.

On the ground of these data, the results of practical experience, it seems that in judging the results of the mallein test, as well as with regard to official regulations, the following principles should be kept in view.

I.—In Case of Suspected Glanders.—It is to be regarded as a reaction when the bodily temperature at the sixth, ninth, eighteenth, or twenty-first hour of observation, from a normal initial temperature, rises above 39° C., and this thermic reaction is associated with an organic one.

The presence of the following clinical symptoms is to be regarded as an organic reaction: A large and painful swelling appears at the point of inoculation within twenty-four hours; not infrequently other swellings as thick as a child's finger proceed from this one; the animal, when led from the place in the eighteenth to twenty-first hour of observation, is stiff, walks with great difficulty (so that although perhaps he was quite unmanageable at the time of the test, it is now necessary to urge him into movement), and limps, more or less, with the fore-limb of the side inoculated. With the rising of the bodily temperature, the depression, apathy, and loss of appetite increase, and the breathing becomes quicker and more difficult. (Quivering of the muscles forms no typical organic reaction.)

Without the organic reaction, the increase of bodily temperature is only to be regarded as a reaction when, with a normal initial temperature, it exceeds 39·5°.

Individuals showing the symptoms of mallein reaction here indicated are to be regarded as glandered, and their immediate destruction is necessary.

On the other hand, the outward symptoms leading to suspicion of disease are not to be regarded as originating in glanders when, in the absence of organic reaction, the thermic reaction, with a normal initial temperature, does not exceed 39·5°.

Such animals as do not react to mallein are to be released from quarantine if the external symptoms indicating disease have disappeared, or if it is established beyond doubt that the external symptoms indicating disease do not originate in glanders.

II. In Case of Suspected Infection.—(*a*) It is not to be regarded as a reaction when, in the absence of organic reaction, the temperature, originally normal, does not exceed 39·5° ; *i.e.*, in the circumstances just mentioned the animal submitted to the mallein test is to be regarded as free from glanders.

(*b*) The reaction is uncertain when, with a normal initial temperature, the thermic reaction exceeds 39·5° C., and does not reach 40° C., the organic reaction, however, not following.

The reaction is also uncertain when the temperature exceeds 39° and does not reach 40°, but at the same time the organic reaction appears.

Finally, the reaction is inconclusive when, with a normal initial temperature of over 38·5°, the increase is over ·5°, but does not rise to 40°, the organic reaction appearing at the same time.

In the cases of uncertain reaction here adduced, the presence of glanders cannot be determined, but is not to be regarded as finally disproved; it is therefore necessary to submit animals giving results of this kind to a second mallein test, under which most animals fail to react at all, or in the rarest cases give the typical reaction. If either the pronounced form of glanders, or the symptoms indicating disease, develop in the period between the two mallein tests, the horses should be at once destroyed.

(*c*) The reaction is atypical when the temperature within nine, twelve, fifteen, or eighteen hours rises from the normal to 40° or over, on a single occasion, but does not at the other times when the temperature is taken exceed the normal, or 39·5° at most.

The atypical reaction is rare, and in cases of its occurrence the presence of glanders may be regarded rather as disproved than as present.

In case of atypical reaction, the animal in question is also to be submitted to a second mallein test.

(*d*) The reaction is typical, when the temperature, from the normal or supernormal, rises to 40° or over, whether the organic reaction has shown itself or not. But the typical reaction is corroborated if the organic one also appears.

The typically reacting animals are to be separated as much as possible from those otherwise reacting, as the former are, in all probability, glandered.

The animals giving the typical reaction are to be submitted to a second mallein test, but should the organic reaction also have appeared their destruction would be advisable after the first test.

In the case mentioned under (*a*), that is, when the animals do not react in any manner under the influence of mallein, they should be released from quarantine immediately after the mallein test, but with the limitation, that they must not be sold into another district until sixty days have elapsed since their release, and then only if the competent veterinarian finds them sound at that time.

The animals mentioned under (*b*) and (*c*) should undergo the mallein test a second time on the thirtieth day after the first test. The animals described under (*b*), sub-division 3, as not definitely reacting, should be separated from those reacting in other ways, and placed in strict confinement to the stable. With regard to those animals which, according to (*b*), sub-divisions 1 and 2, are of uncertain reaction, the authorities can give permission for these, if strictly separated from other horses, to be employed for work in the district.

Horses giving the typical reaction are to be left in strict confinement to the stable till the second mallein test.

Of those animals submitted, on account of uncertain or atypical reactions, to a second mallein test, those which do not in any way react under the influence of mallein are to be released from quarantine, under the sale-restriction during the time specified for the animals mentioned as non-reacting under (*a*). On the other hand, those a second time found to give an uncertain or atypical reaction should be released under the sale-restriction of sixty days, dating from the second mallein test. Those animals which are treated a second time with mallein on account of typical reaction, and are then found to give uncertain or atypical reactions, should be treated in a similar manner; while those found typically reacting a second time should immediately be destroyed.

PULMONARY GLANDERS AND THE OTHER NODULAR LESIONS WHICH MAY BE MISTAKEN FOR IT.

By M. Riegler, Bacteriological Institute of the University of Bucharest.

Pulmonary glanders may exist as a secondary manifestation, but it frequently exists as primary glanders, or as the only manifestation of that malady, in horses which have lived with others which were openly glandered.

This glanders, having an evolution of a slow, insidious or latent character, cannot be suspected, unless use is made of the thermometer, of mallein injections, or the sero-diagnosis.

The proportion of horses suffering from this kind of pulmonary glanders may rise to 30 or 50 per cent. of the effectives of a group of horses.

It may be considered as a primary pulmonary glanders, in view of the sometimes total absence of glanderous lesions in the other parts of the horses' bodies. The evolution of this pulmonary glanders is slower and more benignant than that manifested by chronic glanders; and, as the lesions are very slight, we may suppose that it is curable.

Experiments and observations made up to the present have shown that pulmonary glanders, whether as the only localisation of this malady, or in co-existence with glanderous lesions in other organs, is often produced by infection through the digestive passages. But for the complete elucidation of the question it is to be desired that new researches should be made, employing, for ingestion experiments upon horses, cultures of the bacillus of glanders, such as are obtained from glandered horses; or, better still, using the excreta or other products of these horses, but only in small quantities.

The lesions of pulmonary glanders are many and varied; but the principal and as it were specific lesion is the glanderous nodule, with different aspects, dimensions, and transformations, which often co-exist in the same animal. These lesions undergo, rarely and in a slight degree, the calcareous infiltration. Glanderous lesions in the bronchial glands are most frequently found at the same time as these pulmonary lesions.

The nodular lesions may have resemblances to, or may be confused with, nodules originating from chronic lesions of bronchitis or catarrhal broncholitis, of peribronchitis, or bronchiectasis, by fibrous or fibro-calcareous nodules of pneumonoconiosis, with metastatic centres of pyæmia, with tuberculosis, the metastatic centres of true neoplases, but principally, and sufficiently frequently, with the translucent, fibrous, or fibro-calcareous nodules of parasitic origin. The nature of the lesions may be determined if there exist glanderous lesions in other parts of the body; but if there are none of these, we try whether there do not exist glanderous alterations in the bronchial glands; we make an attentive examination of the characters, localisations, and relations of these lesions with the pulmonary tissue; and, as the surest means, we employ microscopic examination, culture, and inoculations into experimental animals, for glanderous lesions have at times a perfect resemblance to parasitic ones.

Sometimes calcified lesions of the lung are of a glanderous nature. These lesions are sometimes absolutely sterile, but often enough one may succeed in obtaining mild cultures of the glanders bacillus, or positive inoculations into animals. The best way to success lies in the concomitant employment of inoculation into guinea-pigs and microscopic examination.

CANCER IN DOMESTIC ANIMALS.

By Professor Dr OLT, Giessen.

CARCINOMA in domestic animals exactly corresponds histogenetically with that of man. The cancer cells of the primary swelling and the metastases are descendants of a group of epithelia, or of a single epithelial cell, in which an unlimited power of increase, of unknown origin, is developed and transmitted. All metastasis is to be attributed to transplanted cells, which increase without communicating their property of forming swellings to the epithelium in their new environment. The cells in the neighbourhood of the metastases remain rather passive, or perish from interruption of nutrition. Herein lies the essential difference between swellings and infectious metastases. The latter arise from wanderings of the causes of disease, and fulfil their functions on the tissue of the correspondent infected path.

The Cohnheim theory, which refers all tumours to evolutionary causes, has but the smallest bearing on the case of carcinoma. The origin of carcinoma, in a case observed by the author of this Report, was referable to laminated epithelium which, in consequence of embryonic displacement, covered a large surface in the rectum of a horse. This very rare instance does not, however, explain how it happened that a small portion of displaced epithelium had acquired the character of forming tumours.[1]

The influence of age is also in animals a disposing cause towards cancer. There seem to be no difference as regards prevalence between the sexes, or between original and cultivated races. Neither is it known that a tendency towards cancer is hereditary among animals; nor are there any observations available relative to the appearance of epidemic carcinoma, or to temporary variations in the prevalence of cancer.

Those parts of the body most injured by mechanical wounds, such as harness chafings, are not the favourite seats of carcinoma. Neither do lesions of the tissue of the mucous membrane, caused by animal parasites (*Gastrophilus* larvæ, Strongylidæ, Trichocephalidæ, Tæniæ and others) in vast numbers, with their chronic irritation, form, so far as present observations carry us, factors disposing to cancer. Nor have wounds caused by thermic or chemical means any importance in this regard.

Applications of a high degree of heat, for therapeutic purposes or in operations, and branding of the stud marks, do not, as is known from experience, give rise to swellings, any more than cauteries do, or rubbings with applications that cut the skin. The fact that in fifty-two cases of carcinoma of the penis in horses only geldings were affected, is attributed by A. Sticker to the results of imperfect erection, whereby smegma and other dirt collect in the prepuce. It is, however, to be remembered that the number of geldings is beyond all proportion greater than that of stallions. Lubarsch has stated the prevalence of skin-cancer among animals, founded upon clinical observations made at Berlin. Of 784 cases of canine carcinoma, the outer skin (exclusive of the anus, palpebræ, mamma, penis, and scrotum) was affected in 143 = 23·7 per cent. A. Sticker mentions 784 cases of primary carcinoma among dogs, of which 620 = 80·9 per cent. affected the outer covering. Of these there are, nevertheless, 341 cases affecting the milk glands, 8 the eyelids, 89 the anus, and 16 the penis, so that 166 had their seat in the rest of the skin.

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[1] The preparation in question is to be found in the collection of the Veterinary College at Hanover.

From these and from analogous figures it has often been deduced that the skin of animals is peculiarly susceptible to carcinomatosis, and that this matter could be explained by the great number of wounds. Statistics based on clinical investigations do not, however, justify such assumptions, for carcinoma of internal organs is only in exceptional cases the subject of clinical investigation. The author of this report further contests the assumption that the canine mamma and anus are the peculiar seats of carcinoma. With rare exceptions, tumours in these situations are to be attributed to adenoma. The assumption that carcinomatosis of the skin is disproportionately prevalent among animals is not confirmed by examination of the value of available statistical figures; that is shown by the results of *post-mortems* also.

The cell-inclusions in carcinoma among animals show morphologically perfect correspondence with the observations made on human beings; that these formations (the Plimmer and Russel bodies) have no relation to microorganisms is shown by numerous investigations.

The successful inoculations of transplantable tumours in rats, white mice, and dogs are to be regarded as transplantations pure and simple.

Resumé.

1. Experience gives no warrant for believing that wounds, whether the uncommonly numerous ones caused by mechanical means, or those chemically or thermically produced, are a cause disposing towards carcinomatosis in domestic animals.
2. Lesions of tissue and chronic conditions of irritation set up in the mucous membrane of animals by the presence of animal parasites in countless numbers, or of fragments of plants, need not be taken into account for the etiology of carcinoma.
3. There are no facts available to warrant the assumption of the parasitic nature of carcinoma; and the statistical inquiries, especially into cancer in animals, furnish no grounds of probability for the hypothetical supposition of a cancer parasite.
4. The hitherto successful transferences of certain malignant reproductions are to be regarded as transplantations pure and simple.

FALSIFICATION OF MEAT AND OF ALIMENTARY MEAT PRODUCTS; NEW MEANS OF ANALYSIS.

By Dr Martel, Paris.

There are two principal modes of falsification. The products incorporated are of low value, deteriorated, and sometimes even dangerous; or else the material first employed is purposely supplemented with products so as to mask by their presence defects, deteriorations, or profound alterations. The falsifications and frauds may be classed under the principal following heads: (1) employment of colouring matters, natural or artificial; (2) use of antiseptics; (3) addition of substances of low alimentary value; (4) employment of altered products; (5) incorporation of the meat of diseased animals; (6) use of the meat of the horse, dog, or cat, substituted for others of greater commercial value.

The fraud which has assumed great importance with the great extension of hippophagy consists in substituting in sausages horse's flesh for that of other species of animals.

The investigation of frauds necessitates the combined employment of histological, physical, chemical, and bio-chemical methods.

The glycogen research (qualitative analysis) has not given good results in practice. It is to be hoped that physical and chemical methods will be found which will give better results. The evaluation of the specific gravity should never be neglected. The density of the muscle of the horse is much greater than that of other animals. Some even assert that its specific gravity is a third greater than that of beef. The determination of the indices of iodine, or of the point of fusion of the fats, is incapable of furnishing an indication sufficient for all cases. Determination of the weight of the glycogen (Niebel), or of the taurine (Martel), can serve as an indication. When it is a question of analysing products, fresh, or preserved with salt or antiseptics without preliminary cooking. the method of Uhlenhuth, based on the discovery of Tschistowitsch, gives good results.

The bio-chemical method, of precipitating serums, has acquired, owing to the labours of many, a high point of precision. Practice has shown that the musculo-precipitatives (Kuppin, Gröning, Nicolas, and Vallée) are to be preferred. It has been established that prolonged aqueous macerations, without marine salt, are preferable to macerations in a physiological solution (Kuppin, Michaelis, Rostocki, Gröning, Nicolas, and Vallée).

The precipitating serums preserve their activity even after many months of dessication.

The reaction of sero-precipitation is not exactly specific. Many authors (Michaelis, Weichardt and Kister, Weichardt) seek to avoid this inconvenience by dispensing with precipitating agents of the second order.

The considerable activity of the musculo-precipitants obtained leads to the admission of a *unit* capable of answering to the following conditions: *the quantity of* 0·25 *cc., or about* 5 *drops, of precipitating serum, capable of producing immediately a well defined cloudiness when added to* 10 *cc. of an experimental maceration of* 5 *per cent.* (1 *part of flesh to* 19 *of distilled and sterilised water*); *the reaction being effected at laboratory temperature* (16° *to* 18°), *the macerations for injection at* 10 *per cent., or for experiment, being completed in* 5 *hours, at a low temperature, in a sterile vessel, with flesh of animals healthy and well-bled, which flesh is obtained under conditions of purity, and is first congealed and bruised.*

The method of precipitating serum renders incontestable services. It must, however, be recognised that in Paris special circumstances limit its employment. (Horseflesh minced, prepared at the butcher's stall before the customer, penalties of Article 423 of the Penal Code, provided by the ordinance of 6th June 1866, very rarely applied by the tribunals.)

The following conclusions may be submitted:—

1. The amount of precipitating serum rests on the determination of the weight of the quantity of active musculo-precipitants.
2. The musculo-precipitant is estimated in precipitating units after the method proposed above.
3. A commission might be charged with the task of studying the best processes for the differentiation of meats which might escape the precipitating serum method.

VACCINATION AGAINST FOOT-AND-MOUTH DISEASE.

By M. A. LÖFFLER, Professor of the University of Greifswald.

IT has not yet been definitely established that measures of isolation, of surveillance, and disinfection are sufficient to prevent, in the long run, the propagation of foot-and-mouth disease. As regards small-pox among human beings, which may well, on account of its facility of transmission, be compared to foot-and-mouth disease, this object can be attained only by means of compulsory prophylactic vaccination. Consequently, an efficacious process of prophylactic vaccination might be, in equal measure, of inestimable value in the case of foot-and-mouth disease also.

The question of the prophylactic vaccination of pigs and sheep against foot-and-mouth disease has also been practically solved by the preparation of a prophylactic serum of high value. Horses treated with increasing quantities of virulent lymph furnish a serum which, administered in doses of 5 to 20 cc., according to the weight of the animals, preserves them from the malady during a sufficient length of time.

Horse serum is less efficacious for cattle, for it only immunises them for about a fortnight.

In treating cattle with increasing quantities of lymph, an efficacious prophylactic serum is obtained, which, administered in doses of 0·3 cc. per kilogram, renders the cattle immune during a period twice as long The great expense, however, of manufacturing this serum renders it too costly in practice.

A process assuring a cheaper and more lasting prophylaxis has been arrived at by inoculating cattle four or five times, at intervals of eight to ten days, with moderate doses of 15 to 20 cc. of this serum. But, although it only requires 75 to 100 cc. of serum, this process is still too expensive.

A cheap process can only be obtained with what is called "active" immunisation—that is to say, by inoculation with certain quantities of living virus, attenuated as to pathogenic effect.

The attenuation of virulent lymphs has been achieved by means of agents, physical and chemical. At the same time, attempts made with a view to modify the virulence of the lymph, too violent for cattle, by passing it through the bodies of less sensitive animals, such as the full-grown or the sucking pig (as is done with the virus of human smallpox by cultivating it in the bodies of cattle), have given remarkable experimental results. But all these attempts have failed to secure a process of practical utility.

On the other hand, the systematic researches into the effects of mixtures of prophylactic serum and virulent lymph have recently resulted in the discovery of a new process of active immunisation, inoffensive and cheap.

This process is based on the following principles: (1) that mixtures of certain quantities of concentrated serum and virulent lymph are completely inoffensive when injected under the skin; (2) that the injected mixtures produce immunity, but only when a certain proportion is observed between the serum and the lymph; (3) that this immunity appears three to four weeks after the injection, and that it is at first not very pronounced; and (4) that, as a result of injecting increasing quantities of the mixtures aforesaid, this immunity may be rendered very complete, and equal to that acquired after recovery from the disease itself.

The method consists in inoculating a mixture of $\frac{3}{100}$ (0·03) cc. of virulent lymph and 0·5 cc. of concentrated bovine serum. At the end of three to four weeks $\frac{1}{300}$ cc. is inoculated under the skin in the same way; after twelve to fourteen days more, $\frac{1}{100}$ cc.; and after a new interval of twelve to fourteen days, $\frac{1}{25}$ cc. of virulent lymph. Cattle thus treated may be placed among sick cattle without becoming so themselves. When the animals inoculated have received $\frac{1}{100}$ cc. of lymph they are already immune to a considerable extent.

The cost is 30 to 50 pfennig (3¾d. to 6d.) per head of cattle.

The different methods of prophylactic vaccination are all employed in practice, according to circumstances. One should use:—

1. On farms where the epidemic has broken out—

For pigs and sheep—injections of concentrated horse serum; dose, 5 to 20 cc., according to the mixture.

For cattle—injections of concentrated bovine serum; dose, 0·3 cc. per kilogram.

2. On contaminated farms, where no animal has as yet fallen sick: for cattle—

(*a*) Injections of moderate doses, 15 to 20 cc., of concentrated bovine serum, repeated every eight to ten days.

(*b*) The new method.

3. For healthy stocks, but living near contaminated farms—

Either the method of repeated doses of serum or the new method.

4. For healthy stocks into which animals of exterior origin have been introduced—

The method of repeated doses of serum.

For a general prophylactic vaccination, when the epidemic propagates itself in menacing fashion, only the new method need come into question, it being so cheap, and protecting for so long a time.

The vaccinations termed obligatory ought to be extended, because the amount of transmissible infectious virus is considerably augmented by these vaccinations.

The substances necessary for prophylactic vaccinations should be placed by the State *gratuitously* at the service of owners, for the suppression of epidemics is a question of general interest.

RECENT EXPERIENCES AS TO THE INFECTION OF MAN WITH ANIMAL DISEASES (WITH ESPECIAL REGARD TO MEN PURSUING DIFFERENT TRADES).

By Dr E. VON SZEGEDY-MASZAK, Budapest.

THE diseases shown by previous experience to be transmissible from animals to man are the following: tuberculosis, foot-and-mouth disease, anthrax, rabies, glanders, trichinosis, and, according to some recent experiences, swine erysipelas, and botryomycosis. Besides these, there are some other diseases which we observe in man only in isolated cases, and whose importance in human pathology has yet to be proved.

The transmission of tuberculosis from cattle to man is, as we know, admitted as a fact by the majority of investigators. The contrary view is held by Robert Koch and his followers. The question is a weighty one, not only from the point of view of sanitation but also from that of political economy. Experience goes to show that the tuberculosis of man and that of cattle are caused by the same pathogenic germ, for it has been observed in numerous cases that the disease may be transmitted from the animal to man in the following manners:

1. It has been remarked that cow-milkers, and farmers having to do with cattle, fall sick of tuberculosis; 2. tuberculosis may arise from injuries of the

skin coming in contact with the bodies of tuberculous animals during the processes of slaughtering, skinning, cutting up, or dissection; such diseases are acquired by veterinarians, butchers, and those employed in slaughter-houses; 3. consumption of the milk of tuberculous cows, or of the butter prepared therefrom, may set up primary intestinal tuberculosis.

Anthrax is transmitted to man from sick animals—cattle, sheep—or those which have succumbed to the disease, or been slaughtered on account of it; also by parts of these animals which are consumed or otherwise employed. The disease is transmitted from diseased animals to those who tend them, also to shepherds, keepers, drivers, and veterinarians; from the carcases of animals it passes to knackers, butchers, and veterinarians; by means of infected hair to horsehair-weavers, and those employed in brush and paint-brush factories and wool-sorting establishments, as also to weavers and furriers; by means of the skins, tanners and those employed in leather-works are infected.

Rabies is transmitted to man principally by means of the bites of dogs, cats, and wolves, and especially to people who are obliged by their calling or mode of life to pass much time in streets or fields; such as workmen, and day and field labourers. But the disease may also be caused by the saliva of rabid animals coming into contact with injuries to the human body, or when a person thus injured comes into contact with the diseased parts, especially the nerve parts, of rabid animals.

Glanders is mostly transmitted to man by his coming into contact with the infectious parts of animals which are suffering from this disease or have succumbed to it; it is mostly transmitted to people who, owing to their callings, have much to do with such animals or the carcases of such; ostlers, drivers, farmers, veterinarians, and knackers; in rarer cases, soldiers, or butchers who sell horse flesh.

Foot-and-Mouth Disease is principally a cattle disease; the transmission of this disease to man depends strictly upon its prevalence among animals. It is so transmitted by means of occupation with animals and consumption of products—milk, cheese, butter—of sick animals; dairy farmers, cow milkers, those employed in the milk business, and butchers, are those chiefly exposed to catching the disease by touching sick animals or infected objects. The disease is not rare, but as the cases are mostly of a mild description, they are seldom commented upon; weakly persons and infants in arms may, however, succumb, and epidemics with several fatal cases occur.

The pathogenicity of the bacillus of swine erysipelas for man has only been recently observed. Experience shows that no harm arises from consumption of the flesh of diseased animals; it has, however, often been remarked that wounds which have come into contact with diseased animals when being inoculated have developed symptoms of a local inflammation. In the literature of the subject we also find recorded the case of a butcher who, injuring his finger when cutting up a pig which had been slaughtered because of swine erysipelas, fell sick of the same disease.

Botryomycosis is a disease of the horse, and is transmitted from sick horses to such persons as have much to do with them, chiefly ostlers and drivers. Many writers have observed the disease in man also, but most deny that it is an independent infection caused by a specific germ.

Trichinosis is a far from rare disease in several countries, and may appear as an epidemic among animals and men also. A slight epidemic of this kind was observed in Hungary in 1891.

In single isolated cases other animal diseases have also been observed in man, *e.g.*, the psittacosis of the parrot; the part played by this disease in human pathology requires, however, a searching investigation.

ROYAL VETERINARY COLLEGE, LONDON.

LIST OF BURSARIES, MEDALS, HONOUR CERTIFICATES, ETC., 1904-1905.

Coleman Prizes.

Silver Medal	Mr L. Danels.
Bronze Medal	Mr W. T. Brookes.
Certificate of Merit	Mr H. S. Ward.

Centenary Prizes (£20 each).

Class A	Mr F. M. Reynolds.
Class B	Mr H. Tudor Hughes.
Class C	Mr A. W. N. Pillers.
Class D	Mr L. Danels.

Royal Agricultural Society's Medals.

Silver Medal	Mr L. Danels.
Bronze Medal	Mr W. H. Taylor.

Ralli Prizes in Practical Surgery.

First Prize £5 5s.	Mr L. Danels.
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RINGWORM IN DOMESTIC ANIMALS.[1]

By Professor G. NEUMANN, Veterinary School, Toulouse.

(Continued from page 207).

II.—RINGWORM IN THE OX.

The various forms of ringworm in the ox have long been recognised, and have been classified according to their chief clinical appearances. Their parasitic nature was determined in 1857 by Gerlach and Reynal.

The disease is always due to some form of trichophyton. The common form is trichophyton mentagrophytes (Ch. Robin).

In two calves suffering from ringworm of a clearly marked tricophytic character, Bodin found an endo-ectothrix trichophyton with large, irregularly-shaped spores forming curved chains. On culture it appeared more closely related to the group of favus than to that of trichophyton. It was, in fact, a variety of trichophyton verrucosum similar to that of the horse and ass, and, in Bodin's words, produced a form of "favus with trichophytoid lesions."

Galli-Valerio regarded a form of endo-ectothrix trichophyton which he found in a calf showing white, dry, ringworm lesions, as identical with R. Blanchard's trichophyton Sabouraudi—a parasite responsible for 30 per cent. of the cases of ringworm seen in children.

It is possible that these three forms may eventually prove to be one and the same species.

Symptoms.—In bovine animals, which appear to suffer more frequently than any others, the disease rarely extends over the entire surface of the skin, but appears more commonly to affect the lips (in calves), the head, neck, upper parts of the body, and, in rare cases, the lower parts of the limbs.

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[1] Translated from the "Revue Vétérinaire," No. 5, 1905.

The first symptom consists in a slight circular elevation, over which the hair stands erect. The epidermis rapidly undergoes proliferation, and crusts, which adhere more or less closely to one another, form, producing a mass about 2 to 7 mm. in thickness. According to Gerlach, such crusts are thicker on pigmented portions of the skin, and often exhibit a greyish-white, fibrous, starchy appearance. On unpigmented portions of the skin (which are usually thinner) the crust is less dense, and is slightly yellowish in colour.

The lesion extends centrifugally; fresh patches form, and neighbouring patches become fused into areas varying in size from that of a shilling to a five-shilling piece, and occasionally even of a cheese-plate.

The affected hairs break off level with the free surface of the skin, rendering the patches more apparent. White hairs are less affected, and some always remain projecting above the crust, causing the patches, when on a white skin, to retain a certain amount of covering.

At first the crust is closely adherent to the skin, and if forcibly detached exposes the swollen and bleeding dermis. Gradually the centre becomes detached, whilst the periphery, representing a more recent lesion, continues to adhere. The crusts then rest on a thin layer of pus, and the still inflamed dermis is punctured with numerous minute apertures, indicating the position of the roots of detached hairs. The pus lifts the crust; gradually it undergoes dessication and forms superposed layers, which may or may not prove adherent to the parasitic products, and which form a new crust. The latter is purely inflammatory in character, and is left after the fall of the first. It no longer contains any parasites, at least within its deeper layers.

This second crust, in its turn, dries, falls away, or breaks up, leaving a smooth spot, over which the hair again grows, either at once or after a short period of desquamation.

The disease is accompanied by well marked pruritus, more marked at the commencement and towards the end than during the intermediate period, but never so acute as in scabies.

Ringworm may undergo spontaneous cure in from six weeks to three months. It is more obstinate in calves than in adults, and want of grooming tends to increase its duration. If it extends over a large surface of the body the disease may seriously affect the animal's health; the cases described by Macorps prove that where pruritus is violent it greatly retards fattening.

The remarks regarding the cause and prognosis of ringworm in the horse apply equally to the ox, except as above indicated.

Causation.—Speaking generally, ringworm is produced in oxen by causes similar to those acting in the case of the horse.

Inattention to cleanliness was evidently the cause of the outbreak of ringworm in calves collected together at Lyons in 1871 in anticipation of a siege. The occurrence of the disease at certain times of the year, which has often been referred to, may be explained in a similar way. Thus, after affecting cows throughout the winter the disease often disappears in spring. Throughout the winter cows are kept in dark and often filthy sheds, where parasites propagate rapidly, whilst in spring they are sent to grass, where conditions are inimical to contagion.

Spinola attributed to light some influence on the development of the trichophytic patches. He believed that in stalled animals the patches developed on portions of the body directly influenced by light, and that they healed when the parts were sheltered, reappearing on other exposed portions. Gerlach directly denied this, believing the appearances due either to chance or to some other disease having been mistaken for ringworm.

Finally, it has been said that calves are much more liable than adults to contract trichophytic disease.

Ringworm appears to have been observed in all countries, but is much commoner in certain important breeding centres, such as Normandy, La Vendée, Brittany, Oldenburg, Bavaria, England, and Switzerland. It is also common in Australia (G. Fleming).

Sometimes it assumes an epizootic form. Thus around Adelfingen, and particularly in the Commune of Dorlikon (Canton of Zurich), Fehr in 1838 saw an epizootic of trichophyton disease in horned cattle which lasted four years, and extended to a number of human beings, so that eventually most of the inhabitants of Dorlikon were attacked. The description given by the author suggests, however, that the disease was more probably sarcoptic mange. Within three months Macorps (1859) saw ringworm attack more than one hundred oxen in the Canton of Huy (Belgium). Gigard had a similar experience near Lyons. Pflug states that in certain byres the disease is more or less permanent. The same is true of certain pastures.

Contagion.—(1) *From Ox to Ox.* The older practitioners recognised the transmission of ringworm from one bovine animal to another. Carrère stated that the virus of ringworm might persist for a long time in and about byres occupied by calves suffering from the disease if the precaution were omitted of burning tools employed in cleaning the animals, or halters, etc., used to tie them up. This observation was frequently confirmed, and Pflug records the case of a calf suffering from ringworm which transmitted the disease indirectly to all the animals which were subsequently placed in its stall. Gerlach in particular explained such phenomena very clearly and exactly, and supplemented his teaching by numerous and absolutely convincing experiments.

In these experiments the crusts removed from animals suffering from ringworm were divided, and were applied with slight friction to the skin of animals of varying ages. Old subjects proved difficult to inoculate, but animals of from one to two years of age, and particularly calves, very readily took the disease. Inoculation was rendered more certain still by moistening, scratching, or slightly scarifying the skin.

Furthermore, it was noted as somewhat remarkable, that whatever the shape of the inoculated surface—even though it were linear—the lesion always showed a tendency to assume a circular form, and, in fact, appeared as a true circle, unless the seat of inoculation were too extensive. In all cases the eruption appeared in a fortnight.

Gerlach also attempted to re-inoculate parts which had been denuded of hair as a consequence of a primary eruption. The results were always negative, whilst at points where the hair had again grown, either partially or entirely, a new eruption, though of a relatively feeble character, was produced.

To prove that contagion was exclusively due to the fungus, Gerlach inoculated animals with blood obtained by scarifying a ringworm patch, with the blood-stained serosity from beneath a crust, and even with epidermal scales developed after the fall of the crust, but containing no fungi. In none of these cases was inoculation followed by development of the disease.

(2) *Transmission from other Animals to the Ox and vice-versa.*—It has already been mentioned, when dealing with ringworm in the horse, that both Reynal and Nettleship proved the transmission of the disease from horses to calves. Transmission of the disease from the goat to the ox has also been noted, as has transmission from the cat to the horse and to the ox (Williams).

Gerlach succeeded in inoculating the horse from the ox, and Mégnin similarly transmitted the disease from the calf.

Gerlach conveyed the disease from the ox to the dog after slightly scarifying the seat of inoculation. "The fungus growth appeared at the end of from six to eight days. The subject, a young dog, showed violent itching, the skin became swollen and covered with a soft crust, which was followed by a circular bare patch as large as a sixpence or shilling. The hair soon grew again. The crust was then composed of slightly adherent epidermal scales, which, however, contained no fungi." Fenger also observed the transmission of ringworm from the ox to the dog.

Gerlach was unable to inoculate either sheep or pigs from the ox, nor did Railliet succeed in conveying the disease to the rabbit. Nevertheless Railliet declares that in a very few cases the disease has been transmitted to the sheep and to the pig. Similarly, Perroncito records a case of contagion from the ox to the lamb, and Schindelka has seen sheep contract "herpes tonsurans" after being confined in sheds previously occupied by bovine animals suffering from the disease.

(3) *Transmission from the Ox to Man.*—As early as 1820 Ernst described a case of transmission of ringworm from a calf to a girl. Grognier, Köllreuter, Lavergne, Carrère, and Fehr described similar cases. The number of such observations at present existing is very considerable. These cases, most of which refer to calves, lead us to notice the important work of Gerlach, in which the majority are analysed, and in which is given experimental proof of the transmissibility of ringworm from bovine animals to man. Gerlach made numerous inoculations on himself and on several of his pupils with constant success. For fear of producing an intractable form of disease, he never inoculated parts covered by hair, but usually chose the skin of the arm, and these inoculations always produced more or less regularly developed herpes circina within seven or fourteen days after inoculation. Gerlach states that Börensprung also inoculated himself with bovine ringworm and obtained herpes circina.

After Gerlach, Rüffert, Lemaistre, Macorps, Lafosse, Fünfstück, Bergemann, Nettleship, Pflug, Kretschmär, Besnier, Châboux, Gerlier, Boucher, etc., described cases of transmission of ringworm from the calf and ox to man.

Contagion may occur under varying conditions. It is commonest in cowmen, milkers, etc.; the patches generally appear on the wrists and forearms, particularly on the palmar surface, but may occur in any part of the body. Calves with ringworm about the lips inoculate the flanks and udders of the cows they suck. Similarly, the milkers contract ringworm on the hands (particularly on the dorsal surface), wrists, forearms, and also on the forehead and on portions of the head covered with hair by leaning their heads against the animal's flanks. Horand saw a patch of trichophyton disease at the back of the neck in a butcher who had carried on his shoulders a calf suffering from ringworm. Similar observations had previously been made by Cazenave. Finally, according to E. Besnier, herpes circina often occurs on the backs of the hands and on the forearms of butchers who skin young calves. It is evident that the parasite may afterwards be transferred to any other part of the body by contact with the parts primarily affected.

Ringworm may possibly be transmitted to children by the use of clipping machines which have been used on bovine animals.

The form in which the disease occurs in man is that of herpes circina (vesicular herpes, trichophytic vesicular erythema), for the parts attacked are usually those not covered by hair. In parts covered by hair the form is that of tinea tonsurans; on the lips of sycosis. Occasionally the disease affects the eyelashes. Trichophyton disease of animal origin usually exhibits much greater activity than that proper to the new host. The vesicles are more numerous, sometimes of a suppurative character, the pruritus more intense, the patches larger, and the disease more obstinate.

Treatment.—The treatment suggested for the horse applies equally to the ox. Mercurial preparations should be avoided, for animals may be more or less seriously poisoned by licking themselves. In cases of trichophytic disease Pusch recommends successive inunction of each half of the body with 5 per cent. solution of creolin in oil. The animal should be washed two to four days afterwards and the application renewed.

III.—RINGWORM IN THE SHEEP, GOAT, AND PIG.

Little information regarding the dermatomycoses of the sheep, goat, and pig is available.[1] The cases recorded seem to indicate a close resemblance between the symptoms in these animals and those presented by the horse and ox, allowing for modifications due to the nature of the coat.

In sheep suffering from ringworm the wool at first seems matted into small irregular tufts, which grow larger and more numerous and appear felted together at various points. The neck, chest, shoulders, and back exhibit branny crustaceous patches, and the animals suffer from marked pruritus, which causes them to rub and injure the wool.

Ringworm is very obstinate in the goat.

Two pigs described by Siedamgrotzky showed irregular, rounded trichophytic patches from one to two inches in diameter, reddish in tint, without exudation, but covered with abundant scales.

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[1] Kowalewski has described under the head of favus a skin disease which he observed in Tauride, affecting 300 sheep in a flock of 600. The lesions were in the form of dry crusts, whitish or yellowish-grey, situated on the head, and particularly on the jaws, nose, and exterior of the ears. These crusts, which were excavated, and traversed by from two to a dozen hairs, showed after detachment a red, moist surface. The microscope showed in the crusts numerous elements of a fungus. The general health of the sheep was not disturbed. This affection (which exactly recalls sarcoptic mange) was easily cured by very simple treatment. It could not be transmitted to the dog ("Archives Vétér. de Saint Petersbourg," and "Jahresbericht ü. d. Veterinär-Medicin," 1887, p. 94).

In the pig ringworm attacks the croup, the sides of the chest, the flanks, and lateral aspects of the abdomen, but is commonest on the back and on the outside of the quarters. It forms red isolated patches from one to two inches in diameter, covered with miliary vesicles, which are replaced in turn by brown crusts. The bristles remain unchanged and are not shed or broken. There is no pruritus. Contagion from pig to pig occurs readily. The disease may be conveyed from oxen to pigs through the medium of litter removed from cowsheds to piggeries.

Gerlach was unable to inoculate sheep or pigs with ringworm from the ox. Perroncito mentions a case of contagion from the ox to a lamb. Schindelka has seen sheep contract herpes tonsurans as a result of confinement in sheds previously occupied by oxen suffering from the disease. Siedamgrotzky successfully inoculated two pigs and two sheep with ringworm from the horse, and a goat with the disease from the ox. The two pigs infected two others by contact. Contagion from the goat to the ox was noted in the Canton of Zurich in 1852.

In 1876 Lailler communicated to the Société Medicale des Hopitaux a letter written by Lespiau describing an endemic of trichophyton disease seen in the Cantons of Cérét and Arles-sur-Tech; thirty-four persons, of whom twenty-eight were children, were attacked. The disease appears to have been conveyed by a dog, which was first attacked, to a pig (which in these districts often lives in the same house as the human family), and from the pig to man. A moist season appears to have favoured the development of the disease. The fungus particularly attacked the head, eyebrows, cheeks, and genital region, causing lively irritation.

IV.—RINGWORM IN THE DOG.

At the present time four kinds of parasite are known to occur in the dog: (1) Trichophyton caninum (Matruchot and Dassonville); (2) Microsporum Audouini var. caninum (Bodin and Almy); (3) Eidamella spinosa (Matruchot and Dassonville); and (4) Oospora canina (Constantin and Sabrazès). Finally, Sabouraud isolated from a case of multiple ringworm in a dog and from patches of disease (suppurative folliculitis) on a child in the same house a trichophyton the cultures of which were identical with those of trichophyton mentagrophytes of the horse, except that they always showed a slight brownish coloration. This may be the commonest parasite in cases of tonsurans disease in the dog, and it may also be confused with trichophyton caninum.

All these forms produce tonsurans disease, except oospora canina, which produces favus.

Symptoms.—(1) *Trichophytic tonsurans.* This disease, though rare in France, is commoner in Germany, at least in the form of suppurative folliculitis, probably due to trichophyton mentagrophytes and trichophyton caninum. Gerlach and Friedberger regard them as closely resembling the ordinary forms of ringworm of the ox and horse so far as their symptoms are concerned.

The disease usually attacks the head and limbs; most commonly it commences around the lips and eyes. But when the disease is of old standing patches may be found on all parts of the body. In the early stages they are bare, rounded, and well defined; as they increase in number and extent they may overlap, forming irregularly-shaped areas. They soon become covered with crusts, which at first are thin and whitish or pale grey in colour. The pruritus is sometimes trifling but at others well marked. As a consequence of rubbing, the crusts become saturated with blood and serosity, and their colour alters to yellow or brown. The hairs which have not been broken are glued together; wounds form and produce an alteration in the appearance of the disease; under the crusts the skin often appears copper-coloured or reddish-brown, and the swollen hair follicles form numerous small nodules about the size of millet seeds. The patches sometimes project 3 mm. above the surface and appear covered with papules. Spontaneous recovery is rare. Nevertheless inoculation of the dog with trichophyton caninum has in certain cases produced patches which healed spontaneously in a few weeks (Matruchot and Dassonville).

(2) *Microsporic tonsurans.—Appearances.* Oval or round patches from 2 to 5 centimetres in diameter, sometimes forming larger areas with irregular margins by confluence. The patches are dry, greyish in colour, and covered with thin crusts; they exhibit brittle projecting hairs broken off close to the follicle, which may easily be withdrawn with forceps. The part withdrawn is surrounded by a kind of sheath formed of minute spores 2 to 3 μ in diameter, arranged irregularly and unmixed with mycelium, which is to be found within the hair itself. There is little or no pruritus (Bodin and Almy).

(3) *Eidamella tonsurans.*—This form has only once been seen. The parasite forms an intricate mycelial network within the follicle, containing scattered chlamydo-spores from 1 to 5 μ in diameter. Inoculation with cultures was not followed by any organic lesion, only complete depilation over a spot about 1 to 2 centimetres in diameter (Matruchot and Dassonville).

(4) *Favus.*—Favus in the dog was long attributed to the same species as that of man (achorion Schönleinii) but is due to a special parasite—oospora canina. The first clinical observations were made by Saint-Cyr, Trasbot, Siedamgrotzky, and Cadiot.

In puppies at the breast favus often commences in the umbilical region and rapidly spreads over a large area. In Trasbot's case the head was so covered with favus that only a few narrow, irregular lines of hairs were left. A suckling bitch, which was very extensively attacked, gave off a distinct and peculiar smell like that of mouldy cheese, or the special smell observed in man and described by Bazain as "a sickly, repulsive odour, reminding one partly of the smell of mice, partly of cat's urine, mould, or macerating animal substances."

Cadiot saw an eight-years-old dog with four fully developed favi: two on one shoulder, one on the tail, and one at the base of the ear. Each consisted of a circular crust, whitish on the surface but sulphur-yellow internally; the surrounding skin was slightly swollen. On the left flank, on the top of the quarters, and at the base of the tail were other favi dying away; the surface of each was deprived of hair, reddish, and partly covered with slate-coloured crusts containing numerous spores.

The fungus as present in the lesions consisted of fine filaments 2 to 3μ in diameter, and of thicker filaments built up of long chains of round or oval bodies about 5 to 6μ in diameter.

Favus can only be mistaken for old-standing trichophyton lesions. As a rule, the favus cups are excavated, while the centres of the trichophyton crusts are more often raised above the surrounding parts by the discharge which forms beneath and which only reaches to the periphery at a later stage. Furthermore, the favus crusts are almost entirely formed of parasitic elements; the trichophyton crusts contain relatively few of these, but consist principally of epidermal cells and sometimes of dried blood or pus globules.

Causation.—In trichophyton diseases of dogs youth has not the same predisposing influence as in other species of animals. Nevertheless, inoculation seldom succeeds except in young dogs (Horand).

In contrast with trichophyton, favus, according to Saint-Cyr, is a disease of youth.

Trasbot's subject was certainly nine years old, but she had been weakened by gestation, by parturition, and afterwards by suckling, and her health so diminished as to permit the parasite to obtain an entrance. It must also be remembered that the oospora had first attacked the puppies, which were not more than twenty days old, and, from them, the mother, although the latter had from the first been exposed practically to the same danger of contagion. Nevertheless, Cadiot's case was eight years old and free from any predisposing cause.

The oospora canina when inoculated on dogs reproduces the characteristic lesions of primary favus. The disease usually extends beyond the point of application, and may be communicated to other parts of the body. It does not tend to spontaneous recovery.

Gerlach (1859) saw a case of spontaneous transmission of trichophyton disease from dog to dog; Friedberger (1876) brought this about experimentally.

The dog may perhaps contract a form of favus due, not to oospora, but to achorion quinckeanum (Zopf), which is the parasite of favus in mice. Trasbot's case of favus was a ratter which carried the rats she caught to her litter. Anderson and Buchanan saw a favus lesion on the front paw of a dog which was in the habit of catching mice; several of the mice when captured were found to be suffering from the same disease. It is thus easy to explain the occurrence of favus on the nose and paws of domestic carnivora.

Conveyance of the Disease to Man.—Lavergne (1838) speaks of three dogs suffering from herpes which inoculated three pupils who looked after them. Friedberger (1876) reports the transmission of ringworm from a dog to a child and to a servant who often played with it. The eruption assumed the circina form, and in the case of the child attacked the face, in that of the servant the neck. Experiments made by Friedberger on himself and on three students with crusts from the dog and from the child remained negative. Horand had previously reported a similar case and Haas a second. Transmission from the dog to man is favoured by the habit of fondling animals. Fröhner has seen many instances.

The microsporum of the dog is transmissible to man, producing a form of tonsurans very similar to that obstinate variety due to microsporum Audouini; this statement is based on the observations of Mibelli and Bunch.

The same is true of favus. Saint-Cyr contracted the disease when handling dogs used for experiment. Sherwell's case, in which two young girls were inoculated with favus by handling two dogs, seems to have been due to achorion quinckeanum.

Treatment.—The various forms of trichophyton disease in dogs are not very obstinate. They may be treated by one of the methods recommended for horses.

In a case of ringworm which had resisted all ordinary forms of treatment, Popow was completely successful in one week with daily frictions with a mixture of 1 part naphthol and 16 parts diachylon ointment.

The microsporic form, even when extensive, yields to painting with pure tincture of iodine followed by applications of carbolic vaseline.

Saint-Cyr recommends the following treatment for favus: Remove the crusts, taking care not to make the parts bleed; then soak the parts once a day with a more or less concentrated solution of sublimate (from 2 to 10 per cent.). Sometimes the favi re-form; the crust must then be removed a second time, before applying the lotion, just as on the first occasion. As a rule, five to six applications suffice. Occasionally a few more may be required, but the disease never lasts for very long. On account of the toxicity of corrosive sublimate, it is perhaps better to use friction with 1 per cent. or even 2 per cent. nitrate of silver ointment. The application must be thorough to ensure the medicament penetrating. Saint-Cyr recommended this treatment, and Trasbot afterwards used it with success.

V.—RINGWORM IN THE CAT.

The dermatomycoses of the cat include one disease due to trichophyton, one to microsporon, and one to favus.

Trichophyton.—This form of ringworm consists in circular depilations, covered with abundant crusts, and sometimes attaining in three weeks a diameter of 15 to 35 millimetres (Fenger). It is due to trichophyton felinum (R. Blanchard) which was carefully studied by Sabouraud, who failed to discover it in any other species of animal in which he found spontaneous trichophyton disease. The parasite affects the ectothrix mode of growth. The disease is quite fugitive. The fungus produces white cultures closely resembling those of T. mentagrophytes of the horse.

Williams' case seems to prove that this disease of the cat is transmissible to the horse and ox. Fenger states it may be transferred from the cat to the dog, and Zürn that the cat may take it from the dog.

It is also contagious for the human species, in which it almost exclusively affects women and children. In them the T. felinum produces a form of disease resembling tonsurans but commonest on hairless parts. Sabouraud named this "dysidrosiform circinate trichophytosis," or "Bietts vesicular herpes iris."

Fengër claims to have inoculated the cat with the T. tonsurans of man. Horand and Vincens have also been successful in so inoculating young animals.

Microsporosis.—One case of this kind was seen by Morotel, in a cat a few months old; there were three bare patches, about the size of a shilling, covered with abundant dry, greyish crusts. The fungus formed around the hair and in the hair follicle a sheath consisting of little spores, about 2 μ in size, polygonal in shape, and crowded together without definite arrangement.

Favus—Favus in the cat was first mentioned in 1847 in a thesis by Jacquetant, a former "interne" of the Antiquaille of Lyons. Zander again mentioned it in 1858. Ten years later Saint-Cyr published his important work on favus in the domestic animals, and gave a good description of the disease.

The dermatophyte of favus in the cat has not yet been studied in cultures, but, reasoning on the base of its mode of origin, it would seem to be caused by achorion quinckeanum (Zopf), which is the cause of favus in the mouse. It is also possible that some cases are due to achorion Schönleinii of Lebert, which produces favus in man.

Symptoms.—Favus most commonly attacks the ends of the paws and the base of the claws, but it may also affect the umbilicus or sides of the chest. It gradually extends, first to the head, especially the forehead, base of the ears, and centre of the nose, then to the external surface of the thighs and other parts of the body. It forms crusts from 1 to 4 millimetres thick, slightly sticky, and, where fresh, of a sulphur-yellow colour; at a later stage greyish or yellowish-grey. They resemble those of favus in man. . In outline they are almost perfectly circular, their margins are slightly excavated and project a little above the surrounding skin; their centre on the contrary is depressed, giving the crust as a whole the appearance of a saucer or shallow cup. These depressions vary in size between that of a pin's head and a shilling. The degree of projection bears no relation to the diameter of the lesions, and sometimes large favi are almost level with the surface of the skin. The "cupping" is much less marked in those near the base of the nail. As a rule one only finds at this point an irregular mass of broken-down crusts. When growing on a restricted surface the crusts break in on one another, destroying the regular outline and altering the earlier features of the lesions. The free surface often exhibits upright, stiff, decayed hairs which seem to spring from the depths of the growth, and can be withdrawn by the slightest traction. At a later stage these hairs fall away, having been completely loosened by the progress of the disease. "If one removes the crusts with care, one finds the skin beneath thinned, depressed, and as though atrophied by compression; but pliable, not ulcerated, quite dry, sometimes pale and anæmic, more frequently red, irritated, and exhibiting beneath the surface a few greatly dilated blood vessels. Around the crust the skin is distinctly inflamed, red and thickened, and forms a little distinctly projecting ring" (Saint-Cyr). The disease causes trifling itching, and does not appear to have any injurious result on the general health.

The achorion is found in the lesions in the form of numerous rectangular or ovoid cells, 2·5 to 3 μ wide and 3 to 3·5 μ long, connected together into mycelial filaments of varying length, which break up into short approximately rectangular spores.

Favus in the cat is not a very serious disease. Saint-Cyr saw a cat recover in about three months without treatment.

Causation, Contagion.—Achorion seems almost unable to attack any but young cats. Saint-Cyr twice attempted to transmit favus to adults but failed on each occasion, whilst the seven or eight attempts made with cats of six to eight weeks old all succeeded.

Whenever the origin of favus in the cats could clearly be traced, it was found to be due to contagion from the mouse or rat. These rodents are, in fact, frequent sufferers from ringworm, and since Bennett's time a number of confirmatory observations have been made by Draper, Friedreich, R. Tripier, Mollière, etc.

In Lyons favus seems a very common disease of mice, and in them often proves fatal. It usually commences in the ear. It is difficult to prove whether mice become infected from men by crawling about, in, or over human garments infected with the disease. But it is certain that they can convey their form of disease to man.

Anderson described a case of favus in a little girl who had touched mice caught in a trap, and who conveyed the disease to the majority of her brothers and sisters. Some days afterwards five mice were caught; one of them had a favus "cup" on its back close to the tail, another had the sides of the head and the ears ulcerated as a consequence of the disease. R. Tripier of Lyons successfully inoculated himself with ringworm from the mouse. Horand of Lyons relates the case of a lady who caught favus from introducing her hand into a rat-trap containing a favus-infected rat. As a general rule, the transmission of favus from the mouse and rat to man is by the intermediary of one of the domestic animals. The dog and cat usually contract the disease from mice. This was so in the case of Draper's cat, and probably also in all those in which the disease has been observed. Like mice, cats suffering from favus can infect men. In Draper's case a cat gave the disease to two children that played with it. Saint-Cyr, Horand, Anderson, and W. G. Smith have cited instances.

Inversely, the disease may be transmitted from man to the cat. Thus, as early as 1847, Jacquetant remarked, in the thesis already referred to, "In the ward for ringworm in children I saw two cats, with which the little patients played, contract favus, and a form of favus exactly similar to that from which the greater number of the children themselves were suffering,"

Treatment is similar to that of favus in dogs.

THE STRUGGLE WITH TUBERCULOSIS IN DOMESTIC ANIMALS.[1]

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IN Hungary tuberculosis rarely attacks horses or sheep, and, considering the present agricultural conditions, it must also be regarded as uncommon in pigs. Of 207,700 pigs slaughtered in the Budapest slaughter-house during the last two months of the year 1902, 4583, that is 2·2 per cent., were affected with tuberculosis.

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[1] From the "Proceedings of the VIIIth International Veterinary Congress," Budapest, 1905.

The development of the milk industry and the use of the resulting skim milk for the purpose of rearing pigs render an increase of the disease amongst pigs inevitable unless special precautions are taken to prevent it. If only the skim milk and the deposit left in the milk separators were not employed for feeding purposes, or if they were only used after pasteurisation at 80° to 85° C., it would be easy to control this disease in the pig.

Whilst tuberculosis causes no great losses amongst Hungarian horses, sheep, and swine, it is responsible for very considerable losses amongst cattle.

As a consequence of the markedly different agricultural conditions in Hungary, as compared with Western Europe, one would expect tuberculosis to be rare in this country, particularly in animals of the Hungarian breed, and considerable stress has been laid on this view.

Experience, however, has unfortunately proved the falsity of such anticipations, and I am forced to admit that tuberculosis has already made very considerable ravages even amongst Hungarian cattle, and has spread wherever the slightest opportunity occurred.

The last few decades have brought about extensive changes in the agricultural conditions in Hungary, and in the methods of rearing and feeding cattle, the tendency always being to more intense systems of feeding. This fact is due to the increase and development of the milk trade. Wherever the milk business is carried on the tendency is towards stall feeding. The number of animals in one spot increases (in dairies as many animals as possible are placed together in one byre), and this fact is largely responsible for the disease becoming enzöotic. When such farms are stocked with West European milch cattle a centre for the spread of disease is formed, and contaminates Hungarian cattle (which are extensively used by dairymen) equally with other breeds. The records of the Budapest slaughter-house show that amongst animals of West European breed the cows seem specially liable to tuberculosis (16·3 to 17·8 per cent.), whilst the bulls and oxen, whose life is relatively shorter, only show a percentage of 2 to 3 per cent. of tuberculous animals. Amongst Hungarian cattle, however, the percentage of diseased bulls and cows varies between 4 and 6, whilst in oxen it rises as high as 14 to 18 per cent. In Hungary the West European oxen are fattened in their third or fourth year, and at the latest in their eighth year, whilst the Hungarian ox is utilised for draught up to the tenth or even the sixteenth year, and only then fattened. This necessitates the feeding process being continued for several months longer than in the former case, in order to render the carcase suitable for meat. As a consequence of their longer life, Hungarian animals therefore are more exposed to the chance of becoming infected from the dairies, which act as centres for tuberculosis, whilst the longer period required for feeding favours the spread of disease amongst them.

The Western ox is valuable both as a working animal and a meat producer, and, in order to obtain the best price for the meat, is early prepared for slaughter. The Hungarian ox, on the other hand, is used as long as possible for draught purposes, the owner thinking less of its value as a source of meat. This circumstance alone is sufficient to explain why a larger percentage of the numerous Hungarian cattle are tuberculous than of Western cattle, and why, on the other hand, the extensively used Hungarian cows are much less frequently tuberculous than those of Western breeds.

Were the Hungarian cows used like West European animals they would suffer in a similar degree from tuberculosis, as is proved by the results of tuberculin injections carried out in this country. Of thirty cows on a certain Hungarian estate twenty reacted, that is, 66 per cent.; whilst of thirty-four young cattle nine reacted, that is, only 29 per cent.

At the present time we possess very little exact information regarding the extent to which tuberculosis affects young cattle in Hungary, apart from the recent statistics of the Budapest slaughter-house.

In Upper Hungary during the year 1893, when attempts were being made to stamp out pleuro-pneumonia, it was found that, of about 1000 animals slaughtered in the districts of Arva, Nyitra, and Szepes, and examined *post-mortem*, 3·9 to 5·5 per cent. were tuberculous. Of 218 animals slaughtered in the same year in the district of Trencsén, 21 animals, or 9·6 per cent., were tuberculous. In 1894, 2155 animals were slaughtered in the above districts and examined *post-mortem*. Of these 263, or 12·2 per cent., proved to be tuberculous. In 1895, 568 animals were slaughtered and examined *post-mortem* in the districts of Bars, Sáros, and Szepes. Of these 109, equal to 19·2 per cent., proved to be tuberculous.

The yearly veterinary reports show that every year cases of tuberculosis are met with, but, apart from the above-mentioned figures of the Budapest slaughter-house, only those of the similar establishment at Nagyvárad are worth mentioning. During the year 1900, 3429 cattle were slaughtered there, of which 920, or 9·3 per cent., proved to be affected with tuberculosis.

Tuberculin tests have been undertaken to ascertain the condition of the cattle on large estates, but as this method receives no support from the State it necessarily has only been practised to a relatively slight extent. The *Annual Veterinary Report* for 1899 states that of 7148 animals, distributed in 180 farms, 2869, or 40 per cent., reacted. The statistics for 1902 show that amongst 3594 animals, 420, or 4·6 per cent., reacted. 1723 of these were less than two and a half years of age, and yielded 170 reactions, equal to 9·8 per cent. Among 1871 older animals 250, equal to 11·6 per cent., reacted.

The striking differences between the results of 1899 and 1902 show that the examinations hitherto carried out afford very little ground for conclusions regarding the spread of tuberculosis, and that, in order to obtain reliable data, our attention must be directed to the holdings of the wealthy as well as of the poor peasant classes. In this connection experiments carried out by me during 1903 and 1904 in the Trans-Danubian portions of Hungary afford some valuable evidence. 3508 animals, distributed in twenty important holdings, yielded 58·23 per cent. of reactions. Among these, 2013 adult animals yielded 1517 reactions, equal to 75·36 per cent.; while 1495 young animals yielded 526 reactions, equal to 35·18 per cent.

The data relating to large estates in Hungary therefore by no means correspond with those of foreign estates. In order to learn something of the extent of the disease in the small peasant holdings, I undertook some investigations during the winter of 1898 in six parts of the district of Moson. Of 678 animals, 110 (equal to 16·22 per cent.), reacted. Of adult animals 25·7 per cent. reacted, whilst of 177 young cattle only one gave a positive result. The examinations extended to 117 different farms, sixty-four of which, equal to 54·7 per cent., proved to be infected. Although these data only apply to six parts of the district of Moson, I was able to obtain statistics regarding other portions of the Trans-Danubian district. As a consequence of purchases by wealthy owners from small peasant cattle-breeders, I was able to collect further statistics showing that of 957 adult cattle bought in fifty-four separate lots, 102, or 10·65 per cent., reacted. The number or reactions was relatively much less amongst young cattle than amongst adults.

Of 176 animals, bought in six lots, only four, or 2·2 per cent., reacted. When one studies the origin and degree of infection in peasant breeding establishments, it is at once clear that tuberculosis is commonest in those places which are influenced by neighbouring large estates, or in which tuberculosis may have been introduced by purchases of animals cast by the wealthy owners.

Whilst the above figures afford some information regarding the peasant breeding establishments, I wished in the interest of wealthy buyers to obtain some information regarding the degree of infection amongst those breeds of mountain cattle which are most commonly imported. Amongst such imported mountain cattle, which also are usually obtained from peasant breeders, the number of reacting animals is somewhat smaller than amongst Hungarian peasant-bred cattle. Of 648 adult animals only fifty-one, or 7·89 per cent., reacted, and of fifty-two young animals only one, equal to 2 per cent., reacted. The better results obtained with mountain cattle is easily explicable, not so much on the basis of their more natural surroundings as of the circumstance that they come less in contact with the stall-fed and stall-reared cattle of wealthy owners, and therefore are less exposed to the dangers of infection than the animals of Hungarian peasant breeds.

Having regard to the special conditions obtaining in Hungary, I consider it particularly important, in drawing up orders for the purpose of combating the disease, always to bear in mind the extent of tuberculosis amongst peasant-bred animals as opposed to those bred by wealthy owners. In Hungary large dairy estates not only increase in number from year to year, but those already existing develop both as regards the number and quality of the animals held. The number is chiefly increased by purchases from peasant breeders, certain wealthy owners increasing their stocks almost exclusively by purchase. In connection with these purchases (which are frequently necessitated by the inroads of tuberculosis) the owners commit a very grave mistake, because in almost every instance they place the animals obtained in Hungary or from abroad, and, as a rule, from peasant breeders, amongst the old stock, where they soon become infected; whereas, if they were to subject all newly-bought cattle to the tuberculin test and keep non-reacting animals isolated from the old stock, such new animals proved free from the disease would not only live longer and prove more valuable economically, but would, under the special conditions now obtaining, afford a valuable nucleus for the eradication of tuberculosis.

The basis of Bang's protective measures against tuberculosis consists, on the one hand, in segregating the reacting from the non-reacting animals, and, on the other, in artificial rearing of the calves with milk pasteurised at 80° to 85° C. Artificial rearing of calves was common in Denmark before Bang's suggestion, and therefore caused no particular trouble, because large dairy establishments were already provided with apparatus for pasteurising, and almost every farmer delivered his milk to a central dairy from which the necessary pasteurised milk for the calves was returned. In Hungary, however, central dairy establishments are much rarer than in Denmark, and the cost of similar pasteurising apparatus would involve grave difficulties, for which reason it would be impossible to enforce the pasteurisation of skim milk in Hungary. Moreover, the milk from large establishments is more dangerous on account of the greater extent to which such establishments are infected, and this milk is rarely sent to central dairy establishments. In addition, large owners have grave objections to artificial rearing, and do not practice it, so that to suggest this method would result in the owners withdrawing their support from measures for stamping out tuberculosis. Even in places where the owners do not grudge the trouble of artificial rearing, the attendants are so little accustomed to the method that a large percentage of young calves die from diarrhœa, and even if the method is tried for a time as an experiment it always ends in the owner returning to the usual natural methods of rearing.

On the Archducal estate of Mogyaróvár, where 5000 animals are kept, artificial rearing with pasteurised milk was introduced, and at first great enthusiasm prevailed. Separate calf houses were built, but will now only be used until a byre for non-reacting animals can be arranged for. In this establishment the young calves will be reared by the usual, much more simple and more natural, method of leaving them with their own or with foster mothers. Under such circumstances they seldom suffer from diarrhœa.

Considering the special conditions in Hungary, whenever called on to formulate regulations for checking the spread of tuberculosis, I never insisted on artificial rearing, but suggested that the young calves should be confided to the care of cows which had successfully undergone the tuberculin test.

Purchases made by large dairy owners from small peasant breeders have often proved of great benefit in this connection, because the big dairy herds often possessed no healthy cows which could act as foster mothers. Where, however, no purchases were made from without, and no suitable non-reacting cows could be utilised as foster mothers, I preferred to advise that the calf house be placed as far apart as possible from that containing the cows, and that the calves should only be taken to the reacting mothers in order to suck, and should then again be removed from the cows' byre. Where the young animals are carefully isolated from adults the results of tuberculin injections are always very much more favourable. For instance, it is seen from Table I. that on estate No. 16 only 7·89 per cent. of the young animals reacted. On the Pecsenyéd estate, No. 3, 11 per cent. of young animals reacted. On some estates the figure does not exceed 15 to 18 per cent., although the adult cattle on all these estates reacted in very large proportions. Considering that on the estates mentioned nothing whatever was done against tuberculosis, and yet that relatively favourable results were obtained, and bearing in mind the fact that congenital tuberculosis is extremely rare, it seems probable that the mere removal of the calves from the cows' byre soon after birth would lead to even greater improvement in the results, particularly if the calves of reacting cows were given to non-reacting cows to suckle, so that artificial rearing, which is greatly objected to in Hungary, might be avoided. I have carried out this system with success on various estates, and give the following particulars of results obtained in places where the method has been followed for some years.

On the Tormás estate I examined seventy-one half-bred Simmenthaler animals, of which 43·67 per cent reacted. Few foster mothers could be found, so that calves born of reacting cows had to be left with them until the number of non-reacting cows could be increased from amongst the young produced on the estate, and the calves of the reacting cows could then be given to non-reacting animals to suckle. Each of the two establishments, namely, reacting and non-reacting, was provided with a special staff of servants, and the animals, although perforce kept on one farm and even under one roof, were strictly kept apart by means of locked doors. Thanks to the intelligence of the owners, the orders laid down were fairly well carried out, with the result that, despite the examinations being at the somewhat long intervals of nine months, after the fifth examination no more breeding animals reacted. The byres were simple but well found, the cows' byres having concrete cribs and tiled floors, and the calf-houses good wooden cribs and concrete floors. Within three years the whole establishment was cleared of tuberculosis without new purchases being made, and since these protective measures have been in force only two bulls have been purchased to introduce new blood. Both were tested with tuberculin before being purchased, and failed to react.

The second was the Archducal estate of Alcsut, where in 1899 the examination of the pure bred valuable Simmenthal stud was begun, and has been continued at intervals of one year ever since. The young cattle were placed in separate yards; the cows' byre itself is in the centre, and is divided into two sets of stalls, one provided with good concrete cribs and a concrete floor, the other with simpler wooden cribs and a tiled floor. The calves from reacting cows are artificially reared on pasteurised milk, all of Bang's precautions being carefully carried out. Stalls, however, are only disinfected once a year, and the overseer, who is a very intelligent man, controls the management of both sets of byres.

The superintending officials, having seen that the method pursued on the neighbouring estates was much simpler, requested me to take over the further working of protective measures, which I did in 1902. At the time of the fourth test, of 100 animals examined, 35 per cent. (50 per cent. of adults and 28 per cent. of young animals) reacted. This was practically the same result as at the commencement of the protective measures, when 36·17 per cent. of animals reacted. The only alteration I could recommend was that, having regard to the unfavourable results previously obtained, and to the fact that all the animals were stalled in one and the same yard, the tuberculin test should be carried out every half year, and the stalls of the non-reacting animals be disinfected monthly with milk of lime. At the same time I started artificial rearing, but merely as an exceptional measure, because absolutely no foster mothers were to be obtained. Although the results here were far from being as satisfactory as on the other estates (every examination showing from three to four per cent. of reacting animals) the directors were very satisfied with the result, inasmuch as it enabled them within three years to increase the numbers of the non-reacting herd to a point equalling that of the entire herd formerly, despite the fact that they had begun to rear bulls and sold from eight to ten every year, diminishing the non-reacting herd to that extent. During the course of the present year I have had the non-reacting herd placed in a completely isolated set of buildings, and I am of opinion that the results will steadily continue to improve.

On the third (Pecsenyéd) estate I tested during 1899 a half-bred Simmenthal herd and initiated protective measures. Of seventy-three adult animals, fifty-three, equal to 72·6 per cent., reacted; whilst of forty-two young animals which were kept in several fairly large sheds, only eight, equal to 11 per cent., gave positive results. After the first test it was quite easy to separate the reacting from the non-reacting cows, although they were in the same yards, because a small and a large byre were available. Non-reacting animals were placed in the small byre, but, as this did not afford sufficient room, some were bedded down in the ox byre, a part of which was shut off by a door. Artificial feeding was not resorted to. The calves, as before, were left with their mothers, even when the latter reacted, but later on all calves were reared by non-reacting foster mothers. On an average of seven tests made during a period of five years, 2·1 per cent. of the young animals have reacted. The stalls were disinfected at least once a month with milk of lime. Complete isolation was made difficult by the fact that the same milkers had to work in both byres in order that the milking might be completed as rapidly as possible. Bearing in mind the above conditions and facts, I arranged that the non-reacting herd should first be milked and afterwards the reacting herd, after which the milkers seldom returned to the stalls, but went directly home. The herd was examined on three occasions at intervals of nine months, with the result that these simple measures were found to have produced very good results. On the second test only 6 per cent. of the animals, and on the third no animal whatever, was found to react.

The simplicity of the protective measures resulted, however, in a cessation of watchfulness on the part of the farm servants, as a consequence of which serious omissions were made between the third and fourth examinations, nine newly bought cows being placed among the non-reacting animals without having undergone examination. On being tested somewhat later three of these reacted. A further and perhaps more serious error was that of removing the reacting herd, as their numbers increased, to the premises occupied by non-reacting above-mentioned estates, in giving the young calves to foster-mothers and in isolating the non-reacting portion of the herd. This task of isolation was sometimes rendered very difficult on account of the buildings not having been completed, and occasionally Bang's method of dividing a large byre had to be employed. Sometimes the isolation was very imperfectly carried out. Nevertheless, the results were satisfactory, for, taking an average of nine examinations carried out at intervals of six months, the reactions only amounted to 3·3 per cent., and the system has proved so successful that although the herd has grown to three times its original number the disease has not extended.

The eighth estate, namely, that of Vaál and Szt-László, is mostly stocked with Innthal cattle and smaller numbers of Simmenthal cattle. The total number is about 800, and of these 89·4 per cent. reacted. Of the young cattle 60·1 per cent. reacted, so that 76·6 per cent., or three-quarters of the entire number, were tuberculous. The disease was in so advanced a stage that one was obliged to cast from one-quarter to one-third of all the cows yearly, and, as it was impossible to fill the gaps so left with animals bred on the spot, the loss had to be made up by outside purchases. The cows so purchased, however, never gave so much milk as those bred on the spot. The estate therefore suffered loss on the one hand through the lessened yield of milk, and on the other through the continual sales, because the animals so cast were often sold at very low prices.

The farm is in two parts of Vaál and Szt-László. Most of the cows are kept in good stalls, built of concrete, in which, however, they stand heads together. The young animals occupy special sheds provided with simple wooden cribs and rammed clay floors. The tuberculin test is applied every six months. The calves are entrusted to foster-mothers. The concrete stalls are disinfected with sublimate and the clay floors with milk of lime. Success has followed these measures. In 1899, when they were commenced, the non-reacting portion of the herd amounted only to 21 per cent. of the whole, but in 1904 it had risen to 76 per cent. Apart from the increase of the non-reacting animals, the undoubted success of the plan can also be judged of by the numbers of animals cast. During the three years preceding the initiation of protective measures the average value of the animals cast was 214·67 kronen, whilst, although market prices remained at about the same figure, during the third, fourth, and fifth years following the commencement of testing the value rose to 253·32 kronen. Thus from the beginning of the test the average increase in value was 38·65 kronen. After deduction of expenses the animals so sold produced a yearly income of 4000 kronen.

In addition to the above data I have similar statistics relating to other estates, though these apply only to recent years, during which I have practised the tuberculin test and employed the above protective precautions. I therefore consider that it would be somewhat premature to publish them at present, though I make an exception with regard to certain very interesting investigations on the Archducal estate of Magyaróvár.

On account of its enormous dimensions this estate is of special importance, as it contains more than 5000 head of cattle and is probably the largest on the Continent. Here also tuberculosis has made severe inroads, so that, although in all the fifteen districts into which the estate is divided the byres are constructed on the most modern hygienic principles, cows over eight years of age are only occasionally to be found, because the older animals have to be cast on account of tuberculosis. As there seemed some probability of successfully dealing with tuberculosis by Bang's method, and seeing that here, as on other estates, scarcely a single non-reacting animal could be found, further tests were deferred, and efforts were principally directed to saving the young animals by the Danish system. Wherever possible, special rearing byres for calves were erected, and the calves were reared for the first three weeks on their own mothers' milk, afterwards on mixed pasteurised milk. Every six months they were tested with tuberculin, and in general were kept apart as much as possible.

These protective measures were initiated in 1899, with the result that after five years three-fifths of the entire herd when tested with tuberculin proved absolutely free from tuberculosis. The whole of the protective measures have been carried out by the staff of the estate without the assistance of a veterinary surgeon. It is certainly true that some of the arrangements might be criticised by experts, and that an expert, assisted by so intelligent a body of helpers, might have obtained somewhat more favourable results; nevertheless, the success has been of such a character as to deserve the respect even of a specialist.

It should be observed, that, although this estate possessed special arrangements and a specially drilled staff of helpers, together with a farm stocked with non-reacting cows, the artificial mode of rearing, which had been conducted with great regard for detail, was replaced by natural rearing as soon as it became clear that the latter, under existing circumstances, was simpler, cheaper, and more rapidly effective.

Altogether, 10,553 tests were carried out on the artificially reared young animals, usually at six months intervals, and 400 of these, equal to 7·7 per cent. reacted. Of a total number of 7266 tests carried out on young cattle reared by the natural method, positive reactions were obtained in 239 cases, equal to 3·3 per cent. It should however, be stated that on three of the above-mentioned eight estates the tests were carried out at intervals of nine or twelve months. It would therefore seem that as good results may be obtained with this modified form of Bang's method as with his original method.

I have endeavoured to show by the above data the possibility of successfully fighting tuberculosis by a modification of Bang's method suited to Hungarian conditions. I consider that it is one of the first duties of veterinary surgeons to attempt to limit the ravages of tuberculosis on great estates, because this scourge is very wide spread in Hungary, and under the present system these estates form a source of tuberculous infection for the peasant breeding establishments.

Until some more suitable method is devised I believe that Bang's method with the modifications suggested is the most certain of success.

TABLE I.—*Showing the Distribution of Tuberculosis on Large Estates.*

No.	*Tested.*			*Reacted.*						*Breed.*
				Total Numbers.			*Percentage.*			
	Adults.	*Young Animals.*	*Total.*	*Adults.*	*Young Animals.*	*Total.*	*Adults.*	*Young Animals.*	*Total.*	
1.	28	43	71	24	7	31	85·7	16·2	43·67	Simmenthal. Cross-bred.
2.	42	52	94	26	8	34	61·9	15·4	36·17	Simmenthal. Pure bred.
3.	73	72	145	53	8	61	72·6	11·1	42·1	Simmenthal. Cross-bred.
4.	58	55	113	47	37	84	81·3	67·27	74·33	Simmenthal. Cross-bred.
5.	97	73	170	35	13	48	36·0	17·8	28·2	Simmenthal. Cross-bred.
6.	168	79	247	107	15	122	65·0	18·9	49·5	Bonyhad breed.
7.	102	100	202	80	28	108	78·4	28·0	53·5	Bonyhad breed.
8.	461	310	762	403	181	584	87·4	60·1	76·6	Bonyhad and Innthal breed.
9.	69	40	109	59	19	78	85·5	47·5	71·5	Innthal breed.
10.	49	49	98	31	19	50	63·26	38·77	51·02	Bern - Simmenthal breed.
11.	87	69	156	81	34	115	93·1	49·2	73·7	Pure bred Simmenthal and Bonyhad breed.
12.	25	35	60	20	16	36	80·0	45·7	60·0	Pure bred Simmenthal.
13.	18	17	35	10	3	13	55·5	17·6	37·0	Pinzgau breed.
14.	127	63	190	92	13	105	72·44	20·63	55·26	Simmenthal breed.
15.	56	52	108	23	16	39	41·07	30·77	36·12	Simmenthal half-breed.
16.	56	38	94	42	3	45	75·0	7·89	47·87	Allgau breed.
17.	252	176	428	222	65	287	88·1	36·9	67·05	Pinzgau breed.
18.	211	135	346	128	20	148	60·6	14·8	42·77	Simmenthal breed.
19.	19	14	33	19	5	24	100·0	35·7	72·72	Simmenthal half-breed.
20.	15	32	47	15	16	31	100·0	50·0	65·93	Simmenthal half-breed.
	2013	1495	3508	1517	526	2043	75·36	35·18	58·23	

TABLE II.—SHOWING COLLECTIVE RESULTS OF TUBERCULIN TESTS.

Name		Time of First Test.		At the First Test.									Time of Last Test.		At the Last Test.									At the Second Test after Initiation of Protective Measures.									Repetition	
				Number Tested.			Number Reacted.			Percentage Reacted.					Number Tested.			Number Reacted.			Percentage Reacted.			Number Tested.			Number Reacted.			Percentage Reacted.				
of the Estate.	of the Breed.	Year.	Month.	Adults.	Young Animals.	Total.	Adults.	Young Animals.	Total.	Adults.	Young Animals.	Total.	Year.	Month.	Adults.	Young Animals.	Total.	Adults.	Young Animals.	Total.	Adults.	Young Animals.	Total.	Adults.	Young Animals.	Total.	Adults.	Young Animals.	Total.	Adults.	Young Animals.	Total.	of the Test.	Total
Tormás . .	Half-bred Simmenthal	1898	I.	28	43	71	24	7	31	85·7	16·2	43·7	1905	II.	33	43	76	1	—	1	3·3	—	1·3	159	348	507	3	8	11	1·9	2·3	2·2	Every 9 and 12 months	8
Alcsuth . .	Pure bred Simmenthal	1902	IV.	30	70	100	15	20	35	50·0	28·0	35·0	1904	XII.	33	73	106	2	3	5	6·0	4·1	4·7	122	286	408	8	8	16	6·5	2·7	3·9	Every 6 months	5
Pecsenyéd .	Half-bred Simmenthal	1899	IX.	73	72	145	53	8	61	72·6	11·1	42·1	1904	X.	83	105	188	8	2	10	10·3	1·9	5·3	401	516	917	42	11	53	10·4	2·1	5·7	Every 6 and 12 months	7
Szili . . .	„	1898	XII.	58	55	113	47	37	84	81·0	67·3	74·3	1904	VII.	87	159	246	8	1	9	9·2	0·6	3·7	254	435	689	9	13	22	3·5	2·9	3·1	Do.	6
Hédervár . .	„	1898	VII.	97	73	170	35	13	48	36·0	17·8	28·2	1904	VIII.	126	207	333	5	5	10	3·9	2·4	3·0	694	1110	1804	98	40	138	14·1	3·6	7·6	Yearly	6
Tata . . .	Bonyhád . .	1899	IX.	168	79	247	107	15	122	65·0	18·9	49·5	1903	IV.	151	131	282	2	1	3	1·3	0·7	1·1	774	818	1592	30	24	54	3·8	2·9	3·3	Half-yearly	7
Martonvásár .	Bonyhád and Innthal	1900	IX.	102	100	202	80	28	108	78·4	28·0	53·4	1905	I.	173	176	349	8	6	14	4·6	3·4	4·0	975	893	1868	45	29	74	4·5	3·2	3·9	Do.	9
Szt.-László .		1899	III.	442	225	667	398	124	522	90·0	55·1	78·2	1905	III.	286	244	530	10	1	11	3·5	0·4	2·1	2879	2360	5239	174	106	280	7·3	3·7	5·3	Do.	12
	Total .	. .	. .	998	717	1715	759	252	1011	76·1	35·1	59·0			972	1188	2110	44	19	63	4·6	1·7	3·0	5758	7266	13024	409	239	648	7·1	3·3	5·0		

CATTLE PLAGUE IN EGYPT IN 1903-04-05.

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BEFORE referring to the present outbreak of cattle plague in Egypt, I propose to indicate the channels by which I believe cattle plague was introduced into Africa, and to point out the constant danger Egypt runs of re-infection from trading with countries to the east and the south.

The first invasion of Africa during the last twenty years was undoubtedly due to the introduction of cattle either from Arabia through Aden, or to shipments from Bombay to Massowah in the winter of 1887 and 1888, during the war between Italy and Abyssinia. From Massowah the disease rapidly spread over Italian territory, and thence to Abyssinia. We have no definite information as to whether the disease entered the now Anglo-Egyptian Soudan at that time, but if such was the case the losses must have been small, as there were large numbers of cattle in the Soudan when it was occupied by the Anglo-Egyptian Forces a few years later. In 1888-89 an expedition under the late Major Von Wiessmann started for the interior from German East Africa, and the cattle for the expedition were bought from either the ports of Aden or Bombay, although Major Von Wiessmann did his utmost to obtain suitable cattle locally, as he was informed by me before starting of the results of Italian importation of cattle into Massowah. On his return to Egypt he described to me the symptoms of a disease from which his imported cattle suffered, and which in my opinion resembled cattle plague; he further stated that his losses were so great that it materially hampered the movements of his expedition.

Whether the disease spread from German East Africa, or from Abyssinia to Central Africa, and thence on to South Africa, is not known; probably it spread from both centres.

In 1898 the Eastern Soudan was infected with cattle plague by cattle from Italian territory passing down the khors (valleys) and by the river Atbara; and this happened again in the beginning of the present year (1905).

The Anglo-Egyptian Soudan will undoubtedly be exposed to constant risk of infection with cattle plague from its neighbours in the Italian Soudan and Abyssinia until these countries adopt measures which will entail its suppression, as controlling the movements of cattle from one territory to another is an extremely difficult matter where the frontiers are so large and the countries so semi-civilised. In order that the Anglo-Egyptian Soudan may be able to trade with Egypt without being hampered with vexatious restrictions, it will be necessary for them to either induce their neighbours to stamp out cattle plague or to establish a frontier under such conditions that no animals can possibly pass. I hope, for the credit of the two countries, that the former measure will be adopted.

Egypt's permanent danger in trading with the (south) Soudan (Anglo-Egyptian) is not so much for the Soudan itself, where cattle plague occurs from time to time and is apparently controlled, but to the constant source of infection in the Italian and Abyssinian territories, where cattle plague exists in permanence (from which the Soudan is constantly threatened), and might pass on the infection to Egypt before it made itself felt in the Soudan. The far greater danger for Egypt comes, however, from the East, *i.e.*, from Turkey in Asia, where not only cattle plague but also pleuro-pneumonia and anthrax have found a permanent home. It is to this side of the country that the energies of the Government have been constantly directed, and will, I hope, in the future be more successfully carried out, as everything to my mind points to the importation of cattle from Syrian ports as the most probable origin of the present outbreak of cattle plague in Egypt. It may be asked why, if cattle trade with Syrian ports is so dangerous, does the Egyptian Government allow it? The reply must be that Egypt requires a certain amount of imported cattle, as the country cannot quite supply the necessary number for food and labour. If cheap cattle were not imported the fellaheen would probably sell for the butcher their more valuable working animals, which would ultimately produce such a scarcity that the working of land would suffer.

Whether other countries cannot come to the rescue of Egypt, with a slight advance in prices, remains to be seen. Should they do so there is no doubt that a far better quality of meat will be supplied, for the Asia Minor cattle (particularly those from the Bagdad district, which pass through the Euphrates Valley and are finally shipped from Alexandretta to Egyptian ports) are of inferior quality.

Previous to the outbreak of (1903-04-05) cattle plague, the following precautionary measures were adopted: All cattle and sheep arriving from Asia Minor, Russia, and any other countries suspected of cattle plague were landed at a special point and marched about 500 yards along the coast to the Alexandrian Quarantine Station, where they were retained until slaughtered in a special part of the abattoir. All stomachs, heads, feet, etc., of animals slaughtered were boiled, refuse buried, and skins disinfected by being placed for twelve hours in tanks containing a solution of carbolic acid. All butchers and other persons entering this part of the abattoir were provided with special garments in which they worked, and changed before leaving the abattoir, at the same time disinfecting their hands and feet. All meat was carefully inspected by two qualified European veterinary surgeons. In the quarantine stations similar measures were adopted, and, in addition, all the manure and dead animals were burnt.

No suspicious cases of cattle plague were observed in either establishment before or for some time after the disease was declared to exist in Egypt, which was done on 17th June 1903, by Mr Miller, acting Chief Veterinary Inspector. One naturally wondered why, with all the care taken both at the quarantine and at the abattoir, no cases of cattle plague were detected, but the reason was discovered later on, when a better acquaintance with the disease was made. It was then found that Asia Minor cattle, those spoken of as Bagdadli, or coming from the Bagdad district, would often be affected with cattle plague without showing any clinical symptoms, more particularly during the summer. The disease in these could only be detected by a careful examination of the stomachs and intestines, and during the inspection of meat this is rarely done, as the inspector is too fully occupied to have time carefully to open up the stomach and intestines of every animal. Moreover, during life these animals exhibited no symptoms to indicate that the digestive tract was diseased. Some animals retained a few days in quarantine showed fever with diarrhœa, but no deaths occurred; the classical lesions of cattle plague were not observed, and the number of animals indisposed was never sufficient to lead the inspector to suspect the existence of cattle plague. This statement may sound strange from what one reads of the disease, but it is nevertheless a fact that cattle from Asia Minor (Bagdadli) are affected at times with a mild form of cattle plague which is not recognisable in the living animal, as no suspicious clinical symptoms are present. It is only on careful *post-mortem* that the existence of cattle plague can be detected, and this is rendered at times more difficult, as these animals suffer from a form of malarial fever which causes certain lesions in the stomach and intestines. Thus one can easily conceive of meat from infected animals being allowed to pass from the abattoir to the butchers' shops, and meat containing blood, as nearly all meat does, is, I believe, the very essence of cattle plague virus.

Again, the difficulty of controlling and thoroughly disinfecting all persons who have been in contact with imported animals is such that the possibility of some escaping the disinfection is easily understood, and during the last six months before the outbreak of cattle plague occurred imported animals could not pass along the coast to the quarantine station, for, as this isolated route was required for railway purposes, the animals had to make a slight detour inland, and possibly came in contact with other animals, or other animals may have passed over the same ground.

The existence of this disease was first declared on 17th June 1903 in the north of Egypt (Behera Province, near Alexandria), and it continued during the summer to make steady progress in Lower Egypt and Ghiza Province. From this province cattle were taken south by trade route from Bedrashein to Tamai in Fayoum, which province then became infected. Cattle dealers taking the trade route down the Bahr-el-Youssoufy to Lahoun and on to Beni-Souef soon infected that province. The next outbreak in Upper Egypt occurred in Samallout, a butcher having purchased a diseased animal from a travelling dealer who was passing with his animals along the Ibrahimieh Canal. Efforts were made to stop the movements of animals along the Ibrahimieh and Bahr Youssof, but it was of no avail; the disease arrived near Assiout, and from there it proceeded south down the Souhagieh to Girgeh Province, where a case was detected at Souhag amongst cattle arriving from Assiout Moudirieh. Kena did not become infected until the early summer, 1904.

The controlling of the movement of animals, which is of the utmost importance in preventing the spread of animal diseases, was a new form of legislation, and of such magnitude that the authorities were unable to cope with it. It required a trained army of officials, and the people at first did not understand or believe in its importance; but after a year or more of the severest education it was generally believed in, though even now the people often evade the regulations when possible. To enforce regulations which are distasteful is a very difficult task; it is only when they are believed in by the people and these will help in every way that successful results can be obtained.

In suppressing outbreaks of cattle plague regulations such as those now in existence will undoubtedly check the spreading of the disease, and they should be applied over a very extensive area; no interests should be considered, but everything done with the single object of checking the disease.

The first difficulty was to obtain information. This has improved, and cases during the last year and a half were being reported, but often only when several deaths had occurred and the villagers began to be afraid of being found out.

The next difficulty which the Inspector had to encounter was the finding of a piece of land suitable for isolation purposes. If no waste land exists near the village, every site is opposed, as the owner wants it for cultivation purposes, and it is only with the greatest tact that the land is obtained, as no rent is paid, nor do I think it should be, except by the village. In practice I have found it best to inform the Omda that the site selected must be obtained, and leave him to arrange with the proprietor.

The building of a shelter of boos (stalks of Indian corn) occasions another difficulty, although the boos may be found on the roofs of all the houses in the villages; still, with a strong Omda this difficulty disappears.

In my opinion it should be compulsory for every village, when cattle plague or other diseases of cattle break out, to reserve land and erect necessary shelters. This would be no hardship on any person, while it would assist the Government in dealing with the cattle diseases, and undoubtedly save in the village alone the cost of the shelter many times over.

Burying of Animals.—This was always a difficulty, as land was not easy to obtain except when a piece of Government land existed in the village. Owners of land objected, as no payment was made for the land used, and people objected to dig graves for the same reason; but these difficulties were easily met when the Omda had sufficient authority over the villagers to make it clear to them that it was in their interest to have a special burying ground, and that the Government did not intend to pay for the cost of labour or cost of land used.

In future cases of cattle diseases it would be advisable for the Government to insist on every village immediately providing itself with a burial ground for dead animals, and a law should be passed providing for this.

Markets.—One of the most important measures to be adopted in assisting the arrest of the spread of cattle plague is undoubtedly the closing of cattle markets, and keeping them closed for some time after the disease is believed to have been suppressed. They ought to be opened very gradually, where necessary serumising all cattle on payment, and controlling their movements by issuing permits.

On 13th September 1903 I reported on the question, as everybody considered the restrictions imposed worse than the disease. I pointed out that "it resolved itself into a question of helping cattle trade at the expenses of the farmer, or assisting the farmer to stamp out a disease which injured his herds at the expense of the cattle trader and markets holder." Two markets were opened as a tentative measure, and as no harm apparently resulted others were gradually opened. The harm done was not immediately felt, but later on the mistake made was demonstrated beyond a doubt, and every effort was made to recover the lost ground. The opening of the cattle markets not only assisted the spread of the disease, but also had a bad moral effect on the people, as they thought that the danger was over, and that the Government was taking no further interest in the matter.

During the winter months (of 1903-04) it was absolutely necessary to keep open certain markets in Upper Egypt and allow movement of cattle in order to supply Cairo with meat, as no foreign meat was available except to supply Alexandria and Port Said. It then became a question of starving Cairo and carrying out regulations in Upper Egypt, or feeding Cairo and taking the risks of assisting cattle plague to spread.

Slaughtering of Diseased Animals for Food.—In view of the fact that cattle attacked with cattle plague were being slaughtered for food in most of the public abattoirs, the same permission was given to infected villages, as it was considered inadvisable to destroy so large a quantity of food with a consequent loss to the people. This facility to dispose of cattle had undoubtedly a very bad effect, and assisted very materially in spreading the disease from village to village, because as soon as one village in a district was infected it immediately became the centre of attraction to all the butchers in the surrounding villages, who came and bought the cattle plague meat at a cheap rate, taking it back with them, distributing the meat to the villagers and disease to their cattle. During the year 1903-04, more particularly during the winter months, this method of propagating the disease was brought to my notice, and next to diseased cattle travelling I consider the transport of meat one of the most frequent causes of spreading the disease in the villages.

It is not in my opinion a wise measure to allow the killing of diseased animals, and whenever an outbreak of the disease occurs in a village killing of cattle should be stopped in the district unless they can all be carefully *post-mortemed* by a capable veterinary inspector.

Serum.—There is no doubt that the injection of serum when obtainable in sufficient quantities is the very best treatment possible, but at the beginning of an outbreak and for some months afterwards only small quantities are available, as it takes time to make. Animals in contact with diseased and those showing temperatures should, in my opinion, receive a double dose. In Egypt in the summer months a great deal can be done by carefully isolating infected and contact cattle out in the open with a simple shelter against the sun, and thus the serum can be stored for the cold weather when cattle plague assumes a more virulent form.

Recently there have been two or more distinct exceptions to the general efficacy of serum as a means of suppressing outbreaks of cattle plague both in native and imported animals, but all other inoculations done at the same time and with serum prepared at Abbassieh have given excellent results. It is possible that the serum when injected into imported animals does not afford the same protection against cattle plague as when used amongst native herds, more especially if these animals are exposed to adverse conditions, such as travelling long distances with very little food or water, cold weather, etc. A similar occurrence was suspected in animals arriving from the Soudan in March 1904, when, notwithstanding a large dose of serum, some of them showed symptoms of cattle plague; these cases were mixed with a form of malaria. I pointed them out to Professor Koch, who replied that if these animals had not received 150 cc. of serum a month previous he would have been of the same opinion, but he believed that the serum in the doses mentioned would protect cattle against cattle plague. Recent experience has not supported his view, more particularly in imported Cyprus animals.

The first injection of serum was practised near Nahia, in Ghizeh Moudirieh, where a relative of the Omda of the village offered to have his animals injected, and the villagers agreed to have theirs serumised the following week if no bad results occurred. The village was infected with cattle plague and was losing cattle daily. The results were satisfactory, the villagers of Nahia believed in the serum, and after this very little difficulty was experienced in Ghizeh Moudirieh. The serum was then tried in Menoufieh and Galioubieh with equally good results. Some difficulty was experienced at first in Upper Egypt (Beni-Souef), and at the outset it was necessary to guarantee payment of some animals, as the owner strongly believed that death would be the result. However, when it was found that the serum did no harm, but good, its efficacy was soon the talk of the province, and in many instances villagers came requesting to have their cattle serumised. In one village, in order to test whether the serum was poisonous or not, it was suggested with the greatest seriousness that I should drink some of it, and, if there were no bad results, they would allow their cattle to be injected.

Bile.—From a very limited experience I am of the opinion that bile when carefully selected and used with discretion does not communicate cattle plague, but does confer an immunity against this disease. Among nearly 200 cows and calves in Cairo injected with bile taken from cattle plague animals in the Ghizeh Moudirieh, no cases of cattle plague resulted. It is greatly to be regretted that the use of bile could not be continued, but, after the heavy losses from blood and serum injection, the people absolutely refused to allow their animals to be injected, and in many cases strongly objected to their temperatures being taken.

Blood and Serum.—In Egypt this treatment could never be carried out on a large scale in the villages, but it might with great care be applied to herds where competent supervision is available. It would not, I think, be advisable to adopt this treatment in non-infected districts, as it might be the means of introducing cattle plague into them. In carrying out this treatment one must be certain, (1) that the blood is really active, (2) that it does not contain the germs of malaria, (3) that a correct reaction takes place in injected animals, and (4) that all animals to be injected are carefully weighed or their weight correctly estimated, so that correct doses can be given and serum of known strength used.

At first it was almost impossible to convince the villagers that an animal which had had the disease and recovered was of considerable value and should be kept; they much preferred to sell the "salted" animal, as they found him weak for some time after the attack and consequently unsuited for work. Unfortunately large numbers of these salted animals found their way to the abattoirs and were slaughtered. If the large proprietors had acted on the advice frequently offered during the summer of 1903, they might easily, with a very small outlay, have stocked their farms with salted cattle, and thus have saved a large number which were so readily bought up cheap by the village butcher.

Heat and Sun.—These undoubtedly have a modifying effect on the virulence of cattle plague; for animals that contracted the disease during the summer months and were kept in the open with a slight shelter recovered in large numbers. It would further appear that animals contracting the disease out in the open during the summer had a milder form than those stabled. The greatest mortality was experienced in the winter months. Of course, any adverse circumstances would turn the mild form of the disease (the summer form) into a very acute form, as was experienced in the Keneh Moudirieh, where a very large percentage died in the early summer. There the reasons for the rapid spread and high death rate were not far to seek; I inspected the infected district in May 1904, and a more overworked underfed lot of cattle it would be difficult to find. In one day's hard riding I only saw one bullock receiving an allowance of beans; all the other cattle were eating the worst and dirtiest tibn[1] obtainable. The healthy cattle were skeletons, and those affected with cattle plague never had a fair chance to recover. Cattle were all hard at work threshing the corn and cutting up the straw, and many were worked until they were so ill that they could hardly move; they were then placed in the cordon. The disease passed more rapidly through the cattle of the villages in upper Egypt than it has ever done in lower or middle Egypt, and this, I think, is due to the fact that they were more in direct contact. When the Nile was high and the villages were surrounded with water there was very little room for the animals, and when the Nile was low the drinking water was only obtained from wells, morning and evening the animals were herded together, and drank practically out of the same pail or trough.

No cases were observed in sheep or camels.

Cattle Census, Showing Losses of Cattle and Buffaloes.—It has always been considered inadvisable to attempt to collect the statistics of the animal population in Egypt, as it was thought that the owners would fear that it was the intention of the Government, in obtaining this information, to establish an animal tax, and that innumerable difficulties would be raised.

During the outbreak of cattle plague it became necessary that the Omdas of villages should obtain lists showing the number of cattle and buffaloes in their respective villages, and an order was then sent out that all villages should send in their lists to their Moudirs. This was readily done, the Moudirs forwarding these lists to the Ministry of the Interior, and thus an approximate estimate of the animal population was obtained.

The first returns were collected some time in August 1903, when 959,669 cattle and 718,023 buffaloes were shown to exist. Up to that date cattle plague had done very little harm to the country. During January 1904 returns were again obtained, and they showed a shortage of 155,051 cattle and 47,218 buffaloes; in August 1904 there was a further shortage of 199,596 cattle and 25,009 buffaloes, making a total of 354,647 cattle and 72,227 buffaloes.[1]

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[1] Chopped grain.

These returns showed that the Moudiriehs, which were the most badly attacked with cattle plague, had lost most cattle and buffaloes, thus agreeing with the reports of the inspectors of this service. Kena Province showed in six months an increase, until it was attacked, when within six months a diminution of at least 50 per cent. was registered.

It must not, however, be supposed that this shortage was due entirely to deaths from cattle plague, for it was largely increased by the indiscriminate slaughtering of cattle throughout the districts when cattle plague appeared in the neighbourhood. Never during the last eighteen years has meat been eaten so extensively. Unprincipled butchers and dealers were busy going from village to village alarming the people by telling them that cattle plague was in the next village, and that they had better sell their animals. When cattle plague appeared in a village animals were slaughtered at once by their nervous owners, who reasoned that if their cattle were attacked with plague they would be buried and thus they would lose all, while if they were sold and killed they would at least make £3 or £4 off them. Numbers of healthy animals worth £10 were sold for £3 or £4, and even less. The villages have undoubtedly suffered severely by their cattle plague loss, but never in their lives have they enjoyed so much animal food.

The *Official Cattle Plague Returns* show up to date about 100,000 deaths.

In the beginning of 1904 it became apparent that unless some very active measures were adopted the country would be very short of cattle when the outbreak was over—would, in fact, if the towns were to be supplied with meat, and the cultivation to proceed, have insufficient numbers to meet their demands. A Commission, under the presidency of the Adviser of the Ministry of the Interior, and composed of delegates from the Sanitary Department and Agricultural Society, was then appointed by the Government to consider the question.

It was finally recommended that, if possible, quarantine restrictions against importation of cattle into the interior should be modified, so as to allow working cattle to be imported. This was carried out for nearly a year, but very few imports were made, and it became necessary to rescind this order in reference to cattle coming from Asia Minor, owing to the appearance of contagious pleuro-pneumonia amongst them. It was further proposed that a few hundreds of native cattle should be secured and subsequently sold to the fellaheen in districts where they had lost heavily. This was done, most of the cattle being protected by blood and serum inoculation, which resulted in a small loss, I believe under 10 per cent., and that principally among animals which had the disease in them at the time of inoculation, or were suffering from malarial fever, which, in addition to the reaction from cattle plague inoculation, caused their deaths. Owing to the presence of Texas fever and another form of bovine malaria, it was considered advisable, as a tentative measure, to purchase a few foreign animals from districts where a form of malaria (Texas fever) was believed to exist; consequently, cattle were purchased from Italy, Russia (Southern), Hungary, and Roumania.

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[1] Returns are now (August 1905) being collected, and I have every reason to believe that a considerable increase will be registered.

The animals were to be placed under the ordinary conditions of the country, and one could then observe if they were able to withstand the tick fever.

The animals in most cases arrived rather late for the worst attacks of ticks, and it may therefore be suggested that they were not properly exposed to the danger. In any case, the loss from fever was small, and their survival satisfactory, but there was little or no sale for them, the natives not liking their appearance, and apparently being able to do without them. Experience soon demonstrated that the above measures would not suffice to maintain a sufficient number of animals for working purposes, but the working of the land was also helped by machinery, imports of mules, working of camels, donkeys, buffaloes, mules, and horses.

From the commencement of the outbreak attention was directed to importation of cattle for meat, and great facilities were given to meat being sent to the towns in the interior, as it was thought possible to supplant the native supply by competition, and so save the native animals. Previous to the outbreak of cattle plague about 10,000 to 15,000 cattle were imported yearly, and about 120,000 sheep for slaughtering; in 1903 the number increased to about 33,000 cattle and 186,000 sheep. There was also imported about 5500 horses, mules, and donkeys, 46,000 camels, and 470 pigs. There were imported in 1904 about 74,000 cattle, 368,000 sheep, 56,000 camels, 4000 pigs, and 12,000 horses, mules, and donkeys.

In the beginning of 1904 it was found that if more encouragement were given to the foreigners, in time a still greater supply of cattle would be forthcoming, and the following measures were adopted. No native cattle were allowed to be slaughtered without permission in infected districts (it was necessary to declare the whole of Egypt an infected district), and, further, permission was very rarely given, except in cases of old and useless animals, and to buffalo bull calves. The latter are very little use except for food, and, the buffalo having shown considerable immunity against cattle plague, the movement of these animals did not apparently offer the same danger as moving cattle. Besides, butchers in buying calves rarely went themselves or sent their men, but bought through the local dealers, whereas in buying the larger animals they always found it necessary to see the animals themselves, and so the village visited ran the risk of infection from them.

Foreign cattle were entrained and sent direct to the different abattoirs in the country. Now Asia Minor and Soudan cattle are only admitted to the ports and Cairo; Russian, Roumanian, and others to other towns. Sheep are allowed to enter free from Syria after forty-eight hours' observation, being clipped and dipped in an antiseptic wash, as it was thought that this would reduce the risk of introducing anthrax. No sheep of an infected or contaminated lot are allowed to come up country, all being slaughtered in quarantine.

These measures have undoubtedly saved the country an enormous number of cattle which will be suitable for working purposes, and, in addition, they have removed all inducements for butchers buying and moving stock, which has been of the greatest assistance in controlling the disease.

The restrictions are and will remain in force, and are helping the country to recover itself, as a large number of calves are born daily in the country; and, if during two years the people of the towns can be fed from outside, there will be no necessity to interfere with the local animal. Of course, during the importation of Syrian cattle, which frequently results in cattle plague being brought in, the country runs the risk of re-infection; but, as very little movement of native cattle is allowed, and a large number having had the disease, the risk to the country is worth the supply at present. As soon, however, as movement and slaughtering of native cattle is allowed, then the imports of Syrian and other suspicious cattle to the interior must cease.

MALARIAL CATARRHAL FEVER (BLUETONGUE) OF SHEEP IN SOUTH AFRICA.

By JAMES SPREULL, M.R.C.V.S., Government Veterinary Laboratory, Grahamstown, South Africa.

THE following is an account of a disease which I believe to be peculiar to South Africa. My original description of the disease and many experiments in connection therewith appeared in the *Agricultural Journal* of the Cape Colony in April 1902. As I think it has not received the attention which it deserves, I have endeavoured in this article to describe it more fully, and by the addition of further experimental results to bring the subject up-to-date.

So far, I believe, the only account of this disease which has appeared in English professional literature is a note by the Colonial Veterinary Surgeon, Mr Hutcheon, in the *Veterinary Record* of 12th April 1902. Although its clinical aspect was very accurately described at the Cape by Mr Hutcheon as far back as the year 1880 in his Annual Report, it was not till late in 1900 that Mr R. W. Dixon, M.R.C.V.S., under instruction from his chief, began to make investigation into the nature of this disease with a view to the finding of some means of preventive inoculation. When I took over the work from him in May of the following year he had demonstrated its ready inoculability and the very remarkable fact that the blood of a sick sheep could be preserved so as to maintain its virulence for some considerable time. Starting with these data, and without any previous knowledge of the disease, I was soon able to demonstrate many interesting facts which will be set down in due course, but I think it desirable before going further to give a description of the nature, symptons, and other circumstances connected with outbreaks of this disease.

Nomenclature.—The Colonial Veterinary Surgeon has given the name "Malarial Catarrhal Fever" to this disease, and by this name is it well known in the Cape Colony, at any rate, being often styled simply "Fever." Later it will be shown that the word "malarial," as at present understood, is not now applicable to this disease. A very common name, and perhaps the best pending the discovery of the causal microbe itself, is "Bluetongue," which is a translation of the "Blaauwtong" of our Dutch neighbours, amongst whom it is often called "Bekziekte" (mouth-sickness).

Definition.—Bluetongue is an inoculable disease of sheep, characterised usually by fever and many definite symptoms, comprising chiefly lesions of the mouth and feet.

Distribution.—This disease is common in many parts of the Cape Colony, Bechuanaland, the British Protectorate, and Transvaal.

Prevalence.—As Mr Hutcheon long ago pointed out, it is met with both on high and low veldt under the conditions which are recognised as most favourable for the production of South African horse-sickness. Compared with the latter disease, however, it is much more prevalent and widespread, and it occurs regularly every year in certain localities. It is not so common at high altitudes as in low-lying areas, but it is the local elevation which gives the immunity, and not the absolute elevation above sea-level. The "fever" season is well-marked in many districts, and generally lasts from January to April, but it may appear a little earlier or later, depending upon the rainfall. Indeed, humidity seems to have much to do with its production, for during dry seasons the disease is less prevalent and milder in type, and during the recent great drought it failed to make its appearance upon many farms where it is wont to occur. Another circumstance which also bears out the influence of humidity is that it is met with for the most part in kraals situated in valleys or near rivers, dams, or marshy places; and it is a well-known fact that sheep which are kraaled high up on

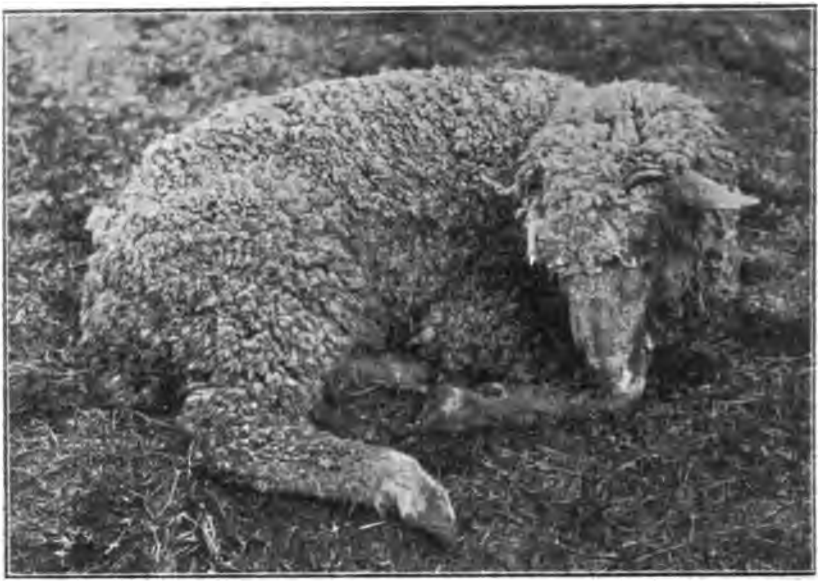

Photo of sheep affected with bluetongue, taken on nineteenth day after inoculation, when the mouth had recovered and cachectic stage ensued. The photo shows a typically twisted neck and also a tendency to shedding of the hoofs. (Photo by Mr T. Bowhill, F.R.C.V.S.)

a hillside rarely become infected. Outbreaks are said to cease after the first frosts.

Mode of Infection.—Since we now know that the disease is easily conveyed by inoculation and not by contact, it becomes necessary for us to explain by what means the infection is propagated naturally. Mr Hutcheon, in his early writings, gave it as his opinion that some biting insect acted as the carrier of bluetongue infection; and, though this opinion has not as yet been proved, all available evidence up till the present points in that direction.

It is a common belief that sheep suffer less from bluetongue when their wool is long than when newly shorn. This is borne out by the fact that in some districts farmers are afraid to shear their flocks in the months when the disease is prevalent. Doubtless heavy losses following upon shearing have led to this experience. It will readily be granted that sheep in this state are more exposed to the attacks of such pests.

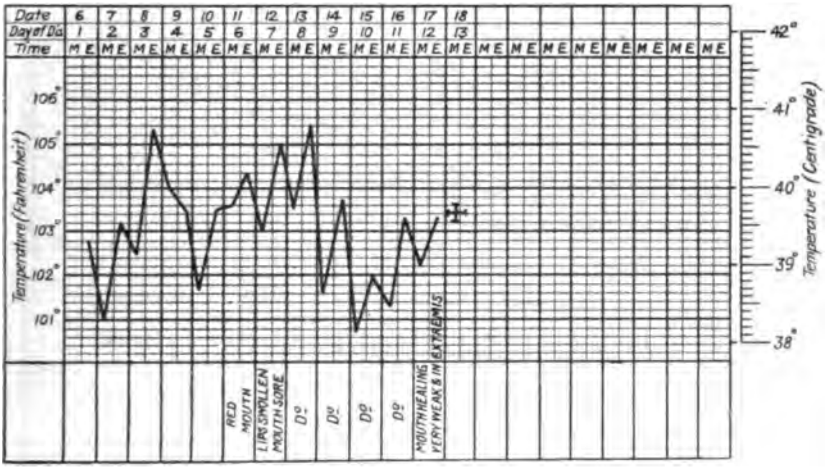

CHART I.

Severe case of sickness followed by death. One-year-old Merino (No. 7), injected intravenously on 6/6/03 with 10 cc. of citrated fresh virulent blood (from No. 2). Died 18/6/03.

Sheep which are placed in sheds at night do not contract the disease. Often a timely change of kraal from a low situation to higher ground will avert an outbreak or check it when it has begun.

Another well-established fact is that dipping in almost any of the dips in common use has a checking influence upon the number of cases in an outbreak, and this we explain by the very natural assumption that the fleece becomes repellent to such insects.

On the same farm certain kraals bear an evil reputation, and in the season it is sufficient to bring sheep from the high ground to such lowly-situated kraals for an outbreak to be speedily produced. On close, warm nights the neighbourhood of these kraals is infested with stinging gnats or mosquitoes, as the visitor will readily find out to his cost. But even if it should yet be proved that an insect is the carrier of the infection, there will remain many interesting points for solution, more especially in regard to the disappearance of the infection in winter and spring-time—facts, no doubt, which are intimately bound up with the life-history of the infection-carrying insect.

Period of Incubation.—The incubatory period of the disease as produced under natural conditions is not known, but experimentally I have found it to be two, three, four, or even five clear days—three and four being the most common, whilst two and five are more rare.

Symptoms.—I shall first describe those manifested in a typical case. The onset of the malady is marked by fever, which is often severe, reaching as high as 107° F.; but temperatures of 104° to 106° are the more usual, and sometimes fever is almost absent, although the symptoms develop in the usual manner and at the proper time. There is nothing very typical about the fever reaction; it is often very erratic, the morning readings especially being lower than one might reasonably expect.

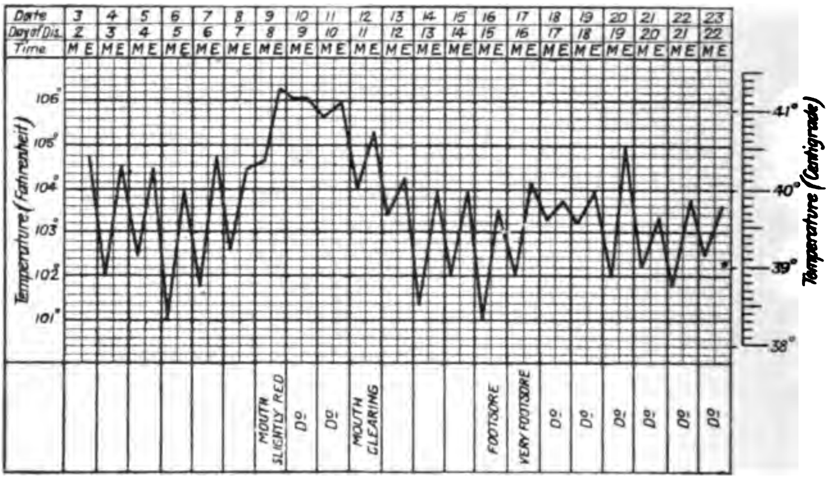

CHART II.

A good case, presenting all the symptoms, and followed by recovery. One-year-old Merino, (No. 198) injected subcutaneously on 2/8/04 with 5 cc. citrated (twenty-two days) virulent blood (from No. 184).

The fever period generally extends from five to seven days, shorter or longer periods being frequent, and there is always a marked drop when the eruption takes place. Although the fever symptoms appear so early, it is not before the seventh to the tenth day that the distinctive lesions in the mouth show themselves. A careful observer, however, would note that the sheep looked thin and did not feed as well as usual for one or two days preceding the eruption. Examination of the mouth may then detect a slight rosiness of the mucous membrane, or occasionally an abnormal blueness, but very often such change is unnoticeable.

The first thing that generally attracts attention is a slight frothing at the mouth and swelling of the lips, particularly the upper lip, with a catarrhal discharge from the nostrils. On examination it will be found that the lining membrane of the mouth is somewhat blue in colour, and the lips may be sore at the junction of the skin and mucous membrane, that is, just along their free margins. Next there is an œdematous infiltration into the mucosa of the dental pad, and also the part immediately behind the incisors; this causes an abnormal whiteness of these parts. In mild cases this condition lasts two or three days, the loosened epidermis is rubbed away by feeding, and recovery rapidly takes place. In a severe case, however, the surface of the pad becomes raw, sores appear inside the lips and cheeks and even on the bars of the hard palate, the tip of the tongue becomes sore by shedding of its epithelium, and may be even gangrenous.

In very severe cases the tongue becomes badly affected, and may be almost wholly stripped of its epithelium and of a dark blue colour; in a few cases this organ becomes extremely œdematous, quite fills the mouth, making swallowing almost impossible, and there is a copious salivary discharge from the mouth. In such cases the tongue, especially on its lateral aspects, where the mucous membrane is thin, presents a glistening yellow or amber colour, resembling a huge blister. In such severe cases the lips become greatly swollen by a serous infiltration into their subcutaneous and submucous tissues, while the nasal discharge, growing profuse, dries upon the nose, diminishes its apertures, and causes difficulty in breathing, manifested by a snoring sound; sores then appear on the skin of the nostrils and muzzle, yet at this stage the nasal chamber seems little affected and is often of a good colour. There is occasionally an œdema of the lower parts of the face, and especially of the skin and tissues under the jaw, extending, though very rarely, for a short distance down the under surface of the neck.

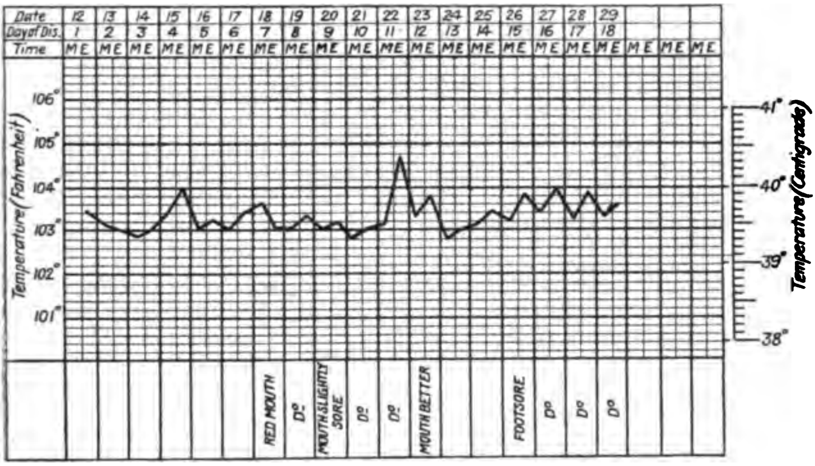

CHART III.

A mild case, practically without fever, yet presenting typical symptoms. One-year-old Merino, (No. 132) injected subcutaneously on 12/2/04 with 5 cc. citrated (six days) virulent blood (from No. 124).

Despite the soreness of the mouth, the sheep have usually an inclination to feed, yet loss of condition is rapid, even when the mouth lesions do not for long interfere with feeding.

Occasionally diarrhœa sets in, and this is always very fatal, blood being passed in the fæces in the later stages. As a rule the fæces are almost normal, except for a more or less abundant slimy mucus discharged along with them.

The mouth lesions generally last from three to five days, but heal less rapidly than they form. Very soon the appetite returns, the feet become tender, and there is a bluish or red line around the coronets, which are swollen and painful on pressure, especially at the heels. It is rare that the skin of the pasterns is inflamed. Most commonly both fore feet are affected; sometimes all four, both hind, or a fore foot and a hind one at the same time.

The severity of the foot lesions is not always in the same degree as the soreness of the mouth. Cases with only slight sore mouth often become very footsore. This symptom generally appears from the fourteenth to the seventeenth day after inoculation, and passes off in a few days, but it is aggravating while it lasts, as it causes the sheep to lie a great deal and refuse to feed; or, if hungry, they feed upon their knees and do not move about enough to fill themselves. The gait of such footsore sheep is very characteristic, and resembles that of a foundered horse. It is only in the worst cases that the feet become tender whilst the mouth is still sore. In some cases the foot trouble is very mild even after marked mouth lesions.

There is great thirst during both the febrile and the eruptive stages. When the sheep begin to feed well again vomiting may supervene, more especially if they have had free access to water. The ingesta are voided by the nose, and in many cases inhaled into the lungs, giving rise to a very fatal pneumonia, which is the only sequel I have met with in this disease.

In young animals, more especially if they be in poor condition, this disease runs a very rapid and fatal course, often with diarrhœic complications. Emaciation is severe, the sheep lie persistently although the feet are not yet affected, the nose is brought round into the flank, and sometimes the neck becomes twisted (wry neck), death ensuing within eight to ten days after inoculation. In such cases the whole skin of the body shows an abnormal rosy hue, the wool can be pulled out in tufts, prostration is extreme, the animal's strength collapses, and after a short period of coma death supervenes. Fortunately such very severe cases are exceptional.

After severe attacks recoveries are very slow, the sheep falling into a state of extreme debility, and the neck at times remaining twisted for a very long time. Forcible reduction of the twist shows that there is neither pain nor stiffness of the part, but sheer muscular atony alone. As a rule the disease has run its course and complete recovery has taken place within tweuty-one days after inoculation. I have never seen the eyes much affected, and shedding of either hoofs or fleece has been very rare and only partial, though I am assured that such symptoms are more frequent and severe in flocks which develop the disease through natural infection.

It will thus be seen that, clinically, the disease may be divided into acute, subacute, and abortive forms. There is no true chronic form.

although death occurs oftener from the resultant debility which follows upon an attack than at the stage when the symptoms are at their height. By inoculation we find that many animals possess a high degree of resistance, and we thus recognise the abortive forms. Such forms range from those showing only an irregular temperature, to those devoloping more or less marked fever reaction, and others still which, in addition to the fever, show for a few days a distinct reddening of the oral mucous membrane.

Mortality.—In outbreaks upon the veldt, as already pointed out, this varies greatly. It may be as little or even less than 5 per cent. of the infected flock, or as high as 30 per cent.; generally, however, with suitable treatment the losses are not very severe. By subcutaneous inoculation I found they ran to 20 per cent., and by intravenous injection they reached 30 per cent., but the number of sheep so treated has not been large enough to enable me to lay this percentage down as absolute. The loss to the farmer, however, is not so much bound up in the number of sheep which actually die as in the great loss of condition which a large percentage of the flock undergoes. There is also a considerable loss in wool, as many of the fleeces become ragged and patchy. Pregnant ewes have been known to abort.

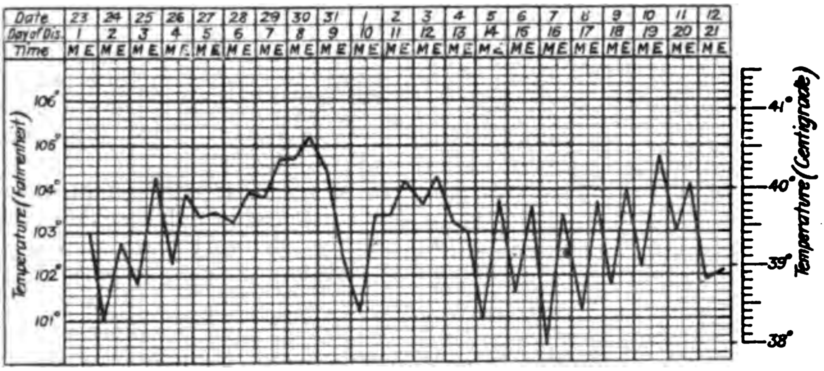

CHART IV.

One-year-old Angora Kapater goat, injected 23/10/03 with 10 cc. citrated fresh blood from goat No. 26 (ninth day in ninth animal of series). This goat shows considerable reaction. The blood taken on the ninth day proved virulent for sheep, showing that the virus of bluetongue had maintained its specific action after passage through ten goats, none of which appeared sick.

Post-mortem Lesions.—As a rule death ensues from debility, and the mouth and other lesions have generally healed by that time. It may happen, however, that the blue line around the coronets is distinguishable, or perhaps an unhealthy-looking wound inside the cheeks and in contact with the molars will assist one to diagnosis. For the rest, all that will be seen is an emaciated carcase.

In severe cases the tongue is stripped of its epithelium and may be gangrenous at the tip. The dental pad and inner surface of the lips are raw and excoriated, and sores are found inside the cheeks in the region of the molars, the latter being usually the last to disappear.

The throat and base of the tongue are healthy, and only in very severe cases do we find the hard palate affected. The œsophagus is healthy, but the rumen often shows inflamed patches, more especially affecting the pillars and the rumeno-reticular margin, and there may even be a slight ulceration at these parts. In cases where vomiting is frequent lesions of the paunch are sometimes lacking on *post-mortem.*

The reticulum is sometimes a little inflamed, but the omasum is almost invariably healthy and its contents soft. The abomasum may be severely inflamed or only mildly so. Sometimes its fundus, and at other times the pyloric end, is most affected, and occasionally the mucous membrane is very œdematous in the latter region. There may be tumefaction and erosions of the rugæ, with one or two ulcers at the pylorus; or, again, the stomach may seem nearly healthy except for a host of punctiform petechiæ, which give its mucous membrane a peppered appearance.

The small bowel is often inflamed throughout, generally most so at the duodenum, and it contains an abundance of sticky mucus. Very often inflammation is only in patches, or there may be slight congestion only. When diarrhœa was severe before death the small intestine is found acutely inflamed with zebra markings, and Peyer's patches are inflamed and prominent. In such cases the large bowel is inflamed even to the rectum, but generally this part of the alimentary canal is much less affected than the small bowel.

The mesenteric lymphatic glands are more or less inflamed and œdematous. The spleen is generally considerably enlarged, soft, and pulpy; it is, however, often firm, and seldom very dark in colour. The liver is congested, but otherwise normal; the bile is normal and the gall-bladder generally full.

The kidneys are congested, and may be marbled on their external surface from hæmorrhage into their cortex; sometimes they are œdematous and the capsules strip off easily. The cortex may be of a brownish colour on section. The bladder is generally full of urine, which is always clear.

In the chest we find a little yellow fluid occasionally, and a little is often present also in the pericardial sac. The heart is usually congested, and in all severe cases shows ecchymoses under its endocardium, and even deeply into the muscular structure; its cavities are generally full of dark, fairly firm clots. The lungs are healthy except when much vomition has taken place; then the dependent parts are found of a dark red colour, hepatised or collapsed, and œdematous, while the trachea and bronchi are full of froth, containing particles of the vomited ingesta, and their mucous membrane is in an acutely inflamed condition.

The subcutaneous tissue of the jaw and under surface of the neck may contain a little colourless or pale yellow watery effusion.

Diagnosis.—The diagnostic lesions are those which are found inside the mouth, and which are unlike those of any other disease. When the season of the year, site of the kraal, and general history of the outbreak are taken into consideration, the diagnosis presents little difficulty. If several cases present themselves for examination, the fever, rapid emaciation, and lameness will complete the picture of this disease in all its phases. The condition known as "Grub in the

Head" is very common in South Africa, but it produces only coughing, sneezing, and a very profuse sticky nasal discharge. Foot-and-mouth disease (epizootic aphtha) would spread more rapidly, and show mainly foot lesions, with nothing in the mouth comparable to bluetongue. The peculiar disease known as geeldikkop is easily distinguished by the swelling of the head, dry gangrene of the ears, and the yellowness of the effusion which exudes on scarifying the skin under the jaw.

Treatment.—For the mouth lesions I have employed chlorate of potash, with tannic and boric acids in glycerine, but such treatment is very empirical and can do little towards promoting the recovery of the parts. Many farmers employ Stockholm tar and sulphur, or aconite and chlorate of potash; others again use tar-derivative sheep dips, and all claim to derive success from these remedies. When the tongue and lips become very œdematous scarification with a fine lancet or needle gives relief. The main idea of all treatments must be to give the sheep complete rest, cool and shady surroundings, and succulent food. When the appetite returns water should be sparingly given, as I think it favours vomiting—a complication which is unusual before recovery has fairly set in. I find that the sick sheep readily lick salt.

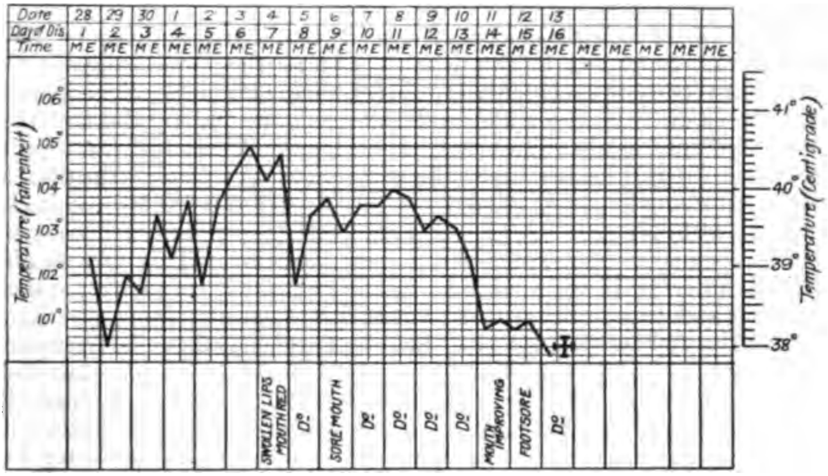

CHART V.

One-year-old Merino wether, injected 28/4/04 with 4 cc. preserved (196 days) citrated blood from goat No. 2 (on the ninth day), which was the eighth in a series. Died 13/5/04. Showed debility only, all lesions of bluetongue having healed.

Prevention.—As already mentioned, it is often possible to check an outbreak by moving the flock to another kraal on a higher situation. By placing the sheep in suitably covered-in sheds the same result will be assured, but these are not often available in South Africa, even for small flocks. Dipping is expensive and often undesirable, so that upon farms where the disease occurs annually some form of preventive inoculation remains as the only means of safety.

Species Affected.—The pure-bred Merino is the most susceptible especially when young; the disease becomes less severe as the adult stage is reached, but there is a great range in the susceptibility of individuals of the same breed. A few Merinos show a nearly complete immunity, others contract abortive forms only. Cross-breds, both of the Afrikander and the Persian, suffer in a considerable degree, but less severely than the Merino. I have not yet had the opportunity of testing a pure Afrikander sheep, but several nearly pure Persians which I tried showed neither fever nor other symptoms, although their blood became virulent after the usual incubatory period.

The goat possesses a strong immunity to bluetongue, and one never hears of a goat developing this disease under natural conditions; further on, however, I shall show that by inoculation the infection can be communicated to this species. In the same way I have infected several calves, though these animals also developed no resultant symptoms. Robertson has attempted to infect the horse and dog, but without success.

Cause of the Disease.—Repeated microscopical examinations have revealed no organism in this disease, but, as the blood conveys the infection readily upon inoculation into a clean animal, we can conclude that the cause is ultra-visible, and this is confirmed by the fact that when virulent blood or its serum only are passed through the Berkefeld filter the filtrate is found to be capable of reproducing the disease (Robertson and Theiler). Mr Dixon and I performed this experiment in 1901 with pippetted serum, the results being exactly similar; we had no means at hand then for proper filtration. This proves also that the microbe is not bound up in the red blood corpuscles, and so we must in future find a better term than "malarial" if we would seek to give the disease a name which will more exactly describe its nature. In many outbreaks of the disease upon the veldt ovine piroplasmosis appears to be associated with bluetongue. In the former disease the piroplasma is readily detected microscopically; it has been known by itself to produce a disease somewhat resembling bovine redwater, but at other times it seems to be but little virulent even when combined with bluetongue.

Modes of Infection.—Bluetongue is not contagious. In my experiments the closest contact of healthy and diseased animals failed to produce it. Even when the drinking water and mangers were soiled with the discharges of the sick animals the in-contact clean sheep remained healthy. In May 1901 Mr Dixon drenched two sheep with 8-10 ounces of virulent blood without producing the sickness, and when I tested them by inoculation some time later I found that they had not acquired immunity therefrom. The blood, uncoagulated, citrated, or defibrinated; blood serum; spleen-pulp and effusions; taken at the acute stages, are all virulent. It seems to matter very little by what means these are inoculated—subcutaneous, intravenous, intrathoracic, and intra-abdominal injections all reproduce the disease alike.

The dose in my hands has not very markedly affected the character of the resulting sickness; in one case a single drop of fresh citrated blood introduced under the skin produced fatal sickness. Some sheep will withstand comparatively large doses of this virus without showing more than a febrile reaction. As a rule, the degree of sickness produced depends more upon the susceptibility of the subject inoculated than upon the apparent virulence of the attack in the sheep from which the blood is taken.

Resistance of the Virus.—Mr Dixon at first tried to preserve the virulent blood by dessication, but in his first attempt the virulence was lost, and so the method was not pursued further. Recently Dr A. Theiler, in the Transvaal, claims to have attenuated blood by dessication, but has not evolved a vaccination method from it yet.

Mr Dixon first demonstrated that the blood could be preserved in the fluid state and still retain its virulence. To the freshly-drawn blood, citrated or defibrinated, he added an equal bulk of a solution composed of equal quantities of glycerine and water containing ½ per cent. of carbolic acid. Blood preserved in this way becomes of a very dark crimson colour. In preserved citrated blood the corpuscles tend to settle down to the bottom of the bottle, but defibrinated blood keeps better, and in it this deposition is less marked. In my hands this blood, kept at the ordinary room temperatures, has proved virulent for very long intervals, though there is an undoubted attenuation after a time. No special care was taken with these samples of blood; they were uncorked frequently when wanted, and the temperature of the room varied in a marked degree according to the state of the weather, yet several different samples, preserved for over 400 days, produced typical symptoms and even death. The longest period I tried was 693 days, and in this case it proved lethal. Preserved blood from a naturally infected case also produced typical sickness and death (in twelve days' time), the blood in this instance being 467 days old. Samples of blood taken from goats also have maintained their virulence on preservation for over 300 days, and possibly do so for even longer periods. Theiler says: "The blood may become putrid and completely decomposed; notwithstanding this, when it is inoculated into a susceptible sheep, bluetongue will result." In order to avoid septic complications he filtered such bloods and injected the filtrate, the disease developing "just as if fresh blood had been injected."

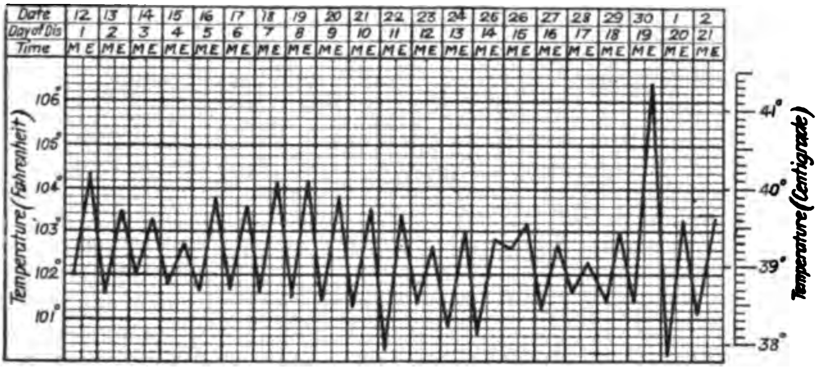

CHART VI.

Eight-months-old calf (No. 1), injected intravenously on 12/6/03 with 10 cc. and subcutaneously with 10 cc. of citrated fresh virulent blood (from sheep No. 7).

In the search for some means of preventive inoculation, it was natural that we should attempt to modify the virulence of the blood of a sick animal by chemical aid. To this end virulent bloods were mixed with solutions of carbolic acid, boric acid, sulphate of quinine, arrhenal, thymol, formalin, and common salt. The mixtures were made up of equal quantities of blood and watery solutions of these drugs of different strengths, and kept at ordinary room temperatures in corked bottles. A carbolic acid solution at 2½ per cent. killed the infection in two samples of blood within twenty hours, whilst in one at 2¼ per cent. the mixture remained virulent for twenty-two days. Weaker solutions of the acid all failed to attenuate, whilst solutions stronger than 2½ per cent. killed the contagium within a few hours. Boric acid at 2 per cent. failed to destroy the virulence of the blood after seven days exposure. A 1 per cent. solution of sulphate of quinine and blood was still virulent on the thirty-first day of exposure in vitro. A 40 per cent. solution of arrhenal did not in the least modify the blood after thirty-eight hours. Thymol was also tried without any marked inhibitory action being noticeable. Mixed with equal quantities of a 5 per cent. solution of common salt, the blood had lost none of its virulence after fifteen days. With ¼ per cent. solution of formalin, after an exposure of sixteen and twenty-five hours a very strange thing happened, for the two sheep inoculated developed sore mouths and typical sickness after sixteen days of an incubatory period. This experiment took place in midwinter, when no spontaneous (fly-infection?) cases could have arisen, and I have seldom since had cases showing such a lengthened incubatory period. After twenty-five days this strength of formalin solution had killed the virulent properties of the blood. A 1 in 1600 solution did not kill the contagium after twenty-four hours' exposure. These experiments are only of value to show how resistant is the contagium of bluetongue. As a means of attenuation they were soon abandoned because of their erratic and discouraging nature; besides, our attenuation was early diverted to the serum methods.

For a time the use of pure glycerine gave some promise of producing the required attenuation, but we had to abandon our efforts in this direction also. When the glycerine and blood were mixed in equal bulk the latter kept its action for ten days or more, but when glycerine predominated in the mixture it speedily became inert. With two of the glycerine mixtures I again had cases developing after very long incubatory periods, viz., fourteen and twenty days. In both cases the glycerine and blood were in equal bulk, and they were in contact in vitro for three and six days respectively. In both cases, as in the two already mentioned, the sickness was severe, and partook more of the nature of a deferred infection than of a mild or attenuated form of the disease.

Bile.—Following upon the lines of rinderpest inoculation, bile from severe cases was injected subcutaneously, either fresh or glycerinated. The fresh bile proved septic, and produced grave sloughing at the point of injection; glycerinated bile, though safer, also caused a good deal of local trouble. No immunity appeared to be conferred by their use, and there is nothing to be said in favour of such injections; moreover, even if they were immunising, it would be impossible to obtain enough of the medium for inoculation purposes.

"*Salted*" *or Recovered Bloods.*—Many injections were performed with the blood of recovered animals, and very varied were the results therefrom. These inoculations showed that the blood of sheep recovering from bluetongue gradually becomes less virulent, but in some subjects this attenuation proceeds much more slowly than in others. In general the blood remained fairly virulent for about thirty days after the fever had abated. Blood obtained from a severe case fifty days after the fall of fever produced typical sickness in a clean sheep which received the dose intravenously, yet in a sheep inoculated under the skin there was no apparent reaction, but this sheep could only have been very imperfectly protected, as it developed a mild sickness after a test performed some time later. On the other hand, the results were somewhat different in the case of another sheep, which contracted rather a mild attack of bluetongue.

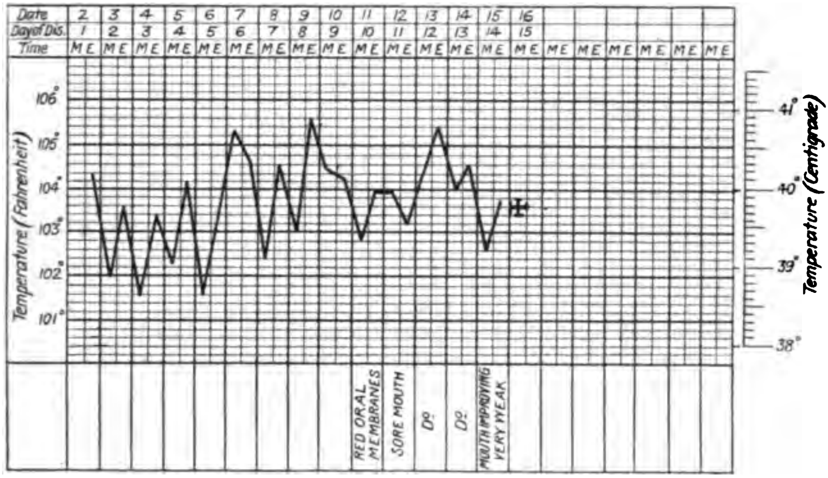

CHART VII.

One-year-old Merino ewe (No. 19), injected intravenously on 2/7/03 with 5 cc. citrated fresh blood from a calf (twenty-one days after injection of blood of sick sheep).

Blood was drawn on the twenty-second day, when the feet were still tender, and it produced mild fever in one case and no reaction in another. When tested, the former showed fever only; the latter developed symptoms and recovered.

All shades of protection have been noted, but blood from sheep over fifty days salted appears to produce little effect. Results were much too irregular for any hard and fast line to be drawn; besides, the individual susceptibility of the sheep thus injected made the results vary greatly, even with the same samples of blood. In a disease like this, where one often finds individuals presenting a strong natural resistance to the contagium, experiments upon a limited number of animals are apt to be misleading.

Preserved Salted Blood.—A number of the samples of "salted" blood used in the above experiments were preserved in the manner already described. These were used upon clean sheep after varying intervals, but they seemed to lose their properties very early. No marked reactions were produced, and there was little evidence of immunity being derived therefrom.

Immunity.—After an attack of bluetongue a sheep develops a very considerable degree of immunity. I was once assured by a farmer that an imported ram of his took the disease every season. I am inclined to doubt the truth of this statement. If sheep were easier to recognise as individuals this point would soon be settled.

If the naturally acquired disease confers no more immunity than the experimentally produced one, then it is far from being absolute. I have frequently produced fever reactions in "salted" sheep by subsequent injections of virulent blood. During such febrile reactions their blood was again found to be active and capable of producing sickness when introduced into fresh sheep. Even when a second injection did not produce fever the blood was sometimes again rendered infective for a time. It often required a large injection before the temperature of a recovered animal was sent up again, and possibly the natural infection would not be strong enough to do this.

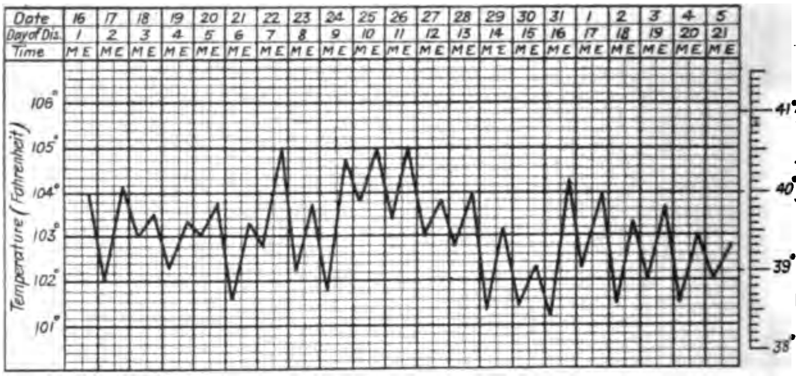

CHART VIII.

A two-tooth Merino wether (No. 280), injected subcutaneously on 16/1/05 with 5 cc. citrated fresh virulent blood (from No. 276) and 3 cc. of serum.

Still later injections are resisted completely. I have only very occasionally induced typical sickness in a "salted" sheep by such severe testing, and in no case did death ensue. Many sheep which passed through the combined serum and blood inoculation without manifesting any symptoms resisted a severe test months later. At Glen Avon the inoculated sheep of one season passed through the next in safety.

Transmission to Goats.—When I first injected virulent blood into a goat no fever reaction ensued, and I was inclined to think that the goat was quite insusceptible; but when I repeated the experiment with a fresh goat, although neither fever nor other symptoms ensued, I removed some blood from it on the twelfth day and injected it into two clean sheep. Both developed typical bluetongue, and one of them died. I have confirmed this observation over and over again, using young Angoras as the subjects of experiment. I found that their blood developed virulence as early as the fifth day, and remained so as late as the twentieth in some cases; but the limit in either direction was not studiously investigated. About the time when the disease would in the case of a sheep be at its height (ninth day) the blood was always highly virulent. When retransferred to sheep at later periods, up to sixteen days, it was very frequently virulent, but I failed to fix any period when a definite amount of attenuation could be counted upon. The blood of these goats, when preserved at its virulent stages, maintained its active properties apparently quite as well as did the sheep blood.

I have also transmitted the contagium of bluetongue through a long chain of goats without giving them more than a febrile reaction, and without apparently either intensifying or attenuating the virus for sheep. At each remove the blood was taken from the goat on the ninth and sixteenth days and injected into susceptible sheep, good attacks of bluetongue invariably resulting therefrom. After ten such removals I brought the experiment to a close. In one goat to which I gave a second injection of the sick blood from a sheep there was no reaction, nor did the blood again prove virulent.

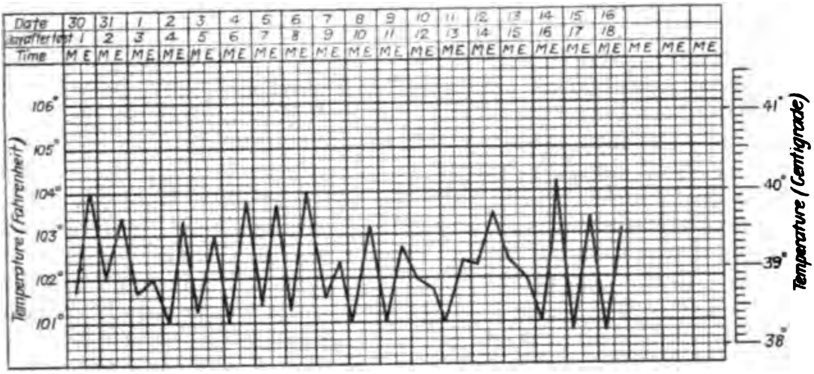

CHART IX.

The same sheep as in Chart VIII., injected subcutaneously on 30/3/05 with 5 cc. preserved (thirty-six days) virulent blood (from Nos. 296 and 299).

Transmission to Cattle.—Although I have had only three animals to work upon, I have nevertheless proved that the contagium of bluetongue can exist in the blood of cattle when transmitted by inoculation. My first, a calf, eight months old, showed a very slight reaction only, yet its blood proved virulent for sheep when drawn on the ninth, thirteenth, and twenty-first days. Out of the five sheep which received this blood, two died; and, curiously enough, one of the latter received the calf's blood of the twenty-first day. Blood taken on the thirty-third day missed two sheep, which afterwards contracted the disease from a test. On the same day the calf was reinoculated with blood from a sick sheep, but its blood did not again become virulent. Eight months later this calf received a fortification injection of 200 cc. of fresh virulent blood, and again there was a mild reaction, but its blood failed to infect a clean sheep on the ninth day. The second calf was of the same age, and no reaction followed upon its injection.

Blood taken on the eleventh day produced reddening of the mouths of two sheep and a slight febrile reaction. On the twentieth day, thinking that an immunity had been acquired, I tested them; both developed severe symptoms after the usual incubatory period, and one died. My third animal, a two-year-old ox, showed no reaction after the injection of sheep blood, and his blood taken on the eleventh day did not convey sickness to the two lambs injected with it. These cattle have since been fortified, but their serum has not proved of much value.

The blood of the first calf was mixed with the preservative solution, and found to keep its virulence for a lengthened period.

Antitoxic Serum.—It has been shown that the blood of sheep recovering from bluetongue gradually loses its infectivity. It was soon apparent to me that a protective action was taking place, so I set about increasing this power by fortifying these sheep with increasingly large doses of fresh virulent blood at regular intervals. It was not long before I found that this method acted just as it had done in the case of rinderpest, and that I was able to produce a serum of strongly protective action. This was in October and November 1901. The testing of the serum was at first conducted only on a small scale within my experimental camp, but it proved efficacious in whatever manner used, provided a sufficient dose was given. Like all serums, it has but a feeble curative action, but, injected either at the same time as a virulent blood or within a day or two afterwards, its controlling power was markedly shown in the character of the ensuing sickness. As a rule, a standard dose of 2 cc. of virulent blood (fresh or preserved) was used, and it was found that the serum diminished its attack in direct proportion to the amount given. Few had more than a mild reaction when 3 cc. was used; with 2 cc. symptoms were frequently developed; whilst 1 cc. was still less effective in checking the advent of severe sickness. In these serum-inoculated sheep it was very interesting to note how abortive forms of the disease manifested themselves; there was every grade of intensity, from those which showed all the symptoms in a mild form, to those in which no reaction whatever was noticeable. Some showed only a blueness or redness of the oral mucous membrane without desquamation, and these frequently became markedly footsore; a few showed the latter symptom alone, sometimes lasting a week, sometimes only for two or three days. Such sheep, although they did not cease to feed, appeared thin and empty after their day's grazing.

It was very shortly after this discovery, or, to be exact, on 10th January 1902, that I was asked to examine some sheep suffering from bluetongue at Glen Avon, which was only four miles from the experimental station at Somerset East. I found three sheep sick in a flock of 295 merinos of all ages. I suggested inoculation, and the owners were good enough to let me try the experiment. Not being over-confident, I bled the sickest of the three sheep and inoculated forty-two only, each receiving two injections, one of blood and one of serum. These did so well that twelve days later I did another lot of fifty, for the disease was spreading and ten fresh cases of sickness had occurred in the flock. On 1st February I inoculated the remainder of the flock, for cases had kept coming on meanwhile. The result was very encouraging; only a few sheep became visibly sick, but mild fever reactions were noted in the majority. Not a single death occurred in the inoculated lot, while ten died among the sheep not treated and about thirty others were sick and recovered.

Our inoculation of to-day differs very little from the method employed in these early experiments. Blood is drawn from highly fortified sheep, and after standing in suitable flasks the serum is collected from it, carbolised, and bottled. It is then ready for use, and will probably keep its potency for many months. The dose I am now using is 4 cc., but 3 cc. is often quite enough to protect susceptible animals. The virulent blood is bottled with the preservative solution, and sent out for use in this very convenient form. It is not necessary that the liquids should be injected separately, and I find it most convenient to mix them immediately before starting to inoculate. By mixing two bottles of serum with one of blood in a suitable vessel one can inoculate rapidly with a dose of 6 cc. of the mixture. The injection should be made under the bare patch of skin just behind the elbow, after previously cleansing the part with some weak disinfectant.

With several "boys" catching and an assistant to fill the syringes (of which it is as well to have two or three in case of accident), it becomes an easy matter to inoculate 100 per hour. I prefer to stand at a table upon which two sheep can be placed at one time; whilst one is being inoculated the other is disinfected, and the latter is then pushed along towards the inoculator, a third taking its place, and so on. For a few minutes after injection the sheep will rear up like a bucking horse, put its nose round to the inoculated side, or perhaps lie down, with other manifestations of pain, but this soon passes off without doing any harm. It is due to the hygroscopic action of the glycerine in the preserved blood.

The effects of the inoculation are generally manifested in from eight to ten days time, when a slight redness of the lining membrane of the mouth will be noticeable. Later on a few sheep will perhaps show a little lameness, but if the serum has been strong little if anything will be noticeable. Sheep under this inoculation therefore require no special treatment.

HEARTWATER AND HORSE-SICKNESS: A NEW PROTECTIVE INOCULATION AGAINST HORSE-SICKNESS.

By J. MORTON COUTTS, M.D. (London), D.P.H. Late Assistant at the Government Bacteriological Institute, Grahamstown, Cape Colony.

IN an article which appeared in this *Journal*, Vol. XVIII., Part 2, June 1905, Messrs Theiler and Stockman criticise a paper by Dr Edington, "On the co-relation of various diseases in stock in South Africa," which appeared in this *Journal*, Vol. XVII., Part 2, June 1904.

I should like to make a few general remarks on the subject, and then give the results of seven cases of inoculation of horses with heartwater, two of which have already been reported in this *Journal*, while a third has been reported in the *Annual Report of the Government Bacteriological Institute*, Grahamstown, Cape Colony, for the half year ending 30th June 1904. The other four cases have not previously been reported.

On first observing the disease heartwater and horse-sickness, the close similarity must strike the observer.

First.—Both diseases can be transmitted by inoculation but neither is infectious.

Second.—Their *post-mortem* phenomena are very much alike.

Third.—They are both peculiar to South Africa.

Fourth.—The contagium existing in the blood is in both cases ultra-microscopical.

These four considerations taken together seemed to me to be too remarkable to be mere coincidences, but rather indicated that the diseases are either identical or have recently diverged from a common parent disease (compare vaccinia). This idea was strengthened on hearing the results obtained by Dr Edington, as reported in his paper "On the co-relation of various diseases in stock in South Africa." During my stay in South Africa those particular experiments were not repeated, and, as I have very little personal experience with regard to them, I shall pass on to the consideration of those which were conducted under my own observation.

During the year July 1903—June 1904, we had found that, if a goat received an intravenous inoculation of heartwater blood on one day, and that this was repeated on the second day, the time of incubation, the length of fever, and the date of death became more constant than if a single inoculation only was given. We also found that only in rare cases did the goat fail to be infected.

It was with this added factor that we now attempted the infection of horses with heartwater with successful results.

In the criticism which appears in the article by Messrs Theiler and Stockman mentioned above, no notice is taken of this double inoculation, and in the report of their experiments mention is made of a single inoculation only of 20 cc., and it is not stated whether this blood was given by the subcutaneous or intravenous method of inoculation. They also threw some doubt on the experiments which have been reported by Dr Edington being correctly interpreted, suggesting that Horse A died of biliary fever, and that Horse B had naturally acquired immunity against horse-sickness.

As regards Horse A, this animal had previously been salted to biliary fever, and this possibly may have given rise to some of the peculiarities found at the *post-mortem*, but the blood of horses suffering from biliary fever does not preserve, and after a period of several months, when inoculated into a clean horse, produce horse-sickness. Yet the blood of Horse A, in doses of 0·25 cc., has produced the disease nearly a year after. Their argument as regards Horse B is one which cannot either be proved or disproved, even when applied to one case; however, it is improbable, and an attempt to extend it to the other cases which have behaved in a similar way would appear ludicrous.

I shall now proceed to report the cases observed by myself at the Institute in Grahamstown. First, however, I shall give the case of an animal inoculated with heartwater during October and November 1901 and January 1902, which has never been reported.

CASE I.—Had the following inoculations:—

24/10/01	20 cc. heartwater blood by subcutaneous inoculation.
28/10/01	20 cc. „ „ „ „
3/11/01	10 cc. „ „ „ „
8/11/01	10 cc. „ „ intravenous inoculation and 200 cc. by subcutaneous inoculation.
2/1/02	10 cc. heartwater blood, intravenous; 30 cc. subcutaneous.
10/1/02	10 cc. „ „ subcutaneous.
12/1/02	5 cc. of virulent horse-sickness blood by subcutaneous inoculation.

I do not give the temperature chart in full, as it is lengthy and without much fluctuation.

On 9th and 10th November 1901 fever reached 102·2°. On 4th, 5th, 6th, and 7th January there was some fever, the maximum being 102·2°. There was a somewhat severe reaction to the large dose of virulent blood, reaching its maximum of 105·8° on 22nd January. The temperature returned to normal on 26th January, the horse recovering.

CASE II.—The animal received the following inoculations:—

17/4/04	10 cc. of heartwater blood by subcutaneous inoculation.
18/4/04	20 cc. „ „ „ „
26/4/04	10 cc. „ „ „ „
30/4/04	10 cc. „ „ „ „

The following gives the temperature record:—

1st day .	— 6 a.m.	9th day .	99·0 6 a.m.
	99·0 noon.		99·4 noon.
	99·4 6 p.m.		100·0 6 p.m.
2nd day .	99·0 6 a.m.	10th day .	99·4 6 a.m.
	100·4 noon.		99·6 noon.
	101·8 6 p.m.		100·8 6 p.m.
3rd day .	100·8 6 a.m.	11th day .	101·2 6 a.m.
	101·0 noon.		101·6 noon.
	101·2 6 p.m.		101·6 6 p.m.
4th day .	99·4 6 a.m.	12th day .	99·0 6 a.m.
	99·0 noon.		100·6 noon.
	101·4 6 p.m.		100·8 6 p.m.
5th day .	99·6 6 a.m.	13th day .	99·4 6 a.m.
	99·6 noon.		99·8 noon.
	101·0 6 p.m.		100·6 6 p.m.
6th day .	99·4 6 a.m.	14th day .	99·6 6 a.m.
	99·6 noon.		100·4 noon.
	100·4 6 p.m.		102·4 6 p.m.
7th day .	99·0 6 a.m.	15th day .	102·6 6 a.m.
	99·8 noon.		103·0 noon.
	100·6 6 p.m.		104·0 6 p.m.
8th day .	99·0 6 a.m.	16th day .	102·0 6 a.m.
	99·4 noon.		103·0 noon.
	100·8 6 p.m.		104·2 6 p.m.

17th day .	103·2 6 a.m.	20th day .	104·0 6 a.m.
	103·6 noon.		104·4 noon.
	104·2 6 p.m.		105·4 6 p.m.
18th day .	103·8 6 a.m.	21st day .	102·8 6 a.m.
	103·8 noon.		102·4 noon.
	105·0 6 p.m.		101·2 6 p.m.
19th day .	102·4 6 a.m.	22nd day .	Dead.
	105·0 noon.		
	105·0 6 p.m.		

CASE III.—The following inoculations were given :—

1/5/05 20 cc. of heartwater blood by subcutaneous inoculation
2/5/05 20 cc. „ „ „ „
9/5/05 20 cc. „ „ „ „
10/5/05 20 cc. „ „ „ „
12/5/05 20 cc. „ „ „ „
14/5/05 40 cc. „ „ „ „
17/5/05 0·25 cc. of virulent horse-sickness blood by subcutaneous inoculation.

The following gives the temperature record :—

1st day .	— 6 a.m.	11th day .	101·0 6 a.m.
	— noon.		101·2 noon.
	101·0 6 p.m.		102·0 6 p.m.
2nd day .	101·6 6 a.m.	12th day .	99·6 6 a.m.
	101·0 noon.		100·0 noon.
	101·6 6 p.m.		102·4 6 p.m.
3rd day .	100·0 6 a.m.	13th day .	99·8 6 a.m.
	100·6 noon.		100·0 noon.
	101·4 6 p.m.		101·0 6 p.m.
4th day .	100·8 6 a.m.	14th day .	100·2 6 a.m.
	99·8 noon.		100·0 noon.
	101·2 6 p.m.		101·8 6 p.m.
5th day .	99·8 6 a.m.	15th day .	100·8 6 a.m.
	99·8 noon.		101·0 noon.
	101·0 6 p.m.		101·4 6 p.m.
6th day .	99·4 6 a.m.	16th day .	100·0 6 a.m.
	100·6 noon.		100·6 noon.
	100·8 6 p.m.		101·0 6 p.m.
7th day .	99·8 6 a.m.	17th day .	100·0 6 a.m.
	100·0 noon.		99·8 noon.
	100·6 6 p.m.		101·0 6 p.m.
8th day .	99·0 6 a.m.	18th day .	100·0 6 a.m.
	100·8 noon.		100·0 noon.
	101·2 6 p.m.		101·0 6 p.m.
9th day .	99·4 6 a.m.	19th day .	100·2 6 a.m.
	100·6 noon.		100·4 noon.
	101·8 6 p.m.		100·6 6 p.m.
10th day .	100·0 6 a.m.	20th day .	100·2 6 a.m.
	101·4 noon.		99·8 noon.
	102·8 6 p.m.		102·0 6 p.m.

21st day .	100·0 6 a.m.	31st day .	104·4 6 a.m.
	99·6 noon.		104·2 noon.
	100·4 6 p.m.		105·0 6 p.m.
22nd day .	100·6 6 a.m.	32nd day .	104·8 6 a.m.
	99·8 noon.		103·8 noon.
	100·8 6 p.m.		104·4 6 p.m.
23rd day .	99·8 6 a.m.	33rd day .	102·6 6 a.m.
	100·0 noon.		103·0 noon.
	101·0 6 p.m.		103·6 6 p.m.
24th day .	99·4 6 a.m.	34th day .	103·8 6 a.m.
	100·2 noon.		101·6 noon.
	101·4 6 p.m.		103·4 6 p.m.
25th day .	100·4 6 a.m.	35th day .	102·0 6 a.m.
	101·0 noon.		102·6 noon.
	102·0 6 p.m.		103·2 6 p.m.
26th day .	101·0 6 a.m.	36th day .	102·0 6 a.m.
	100·8 noon.		101·0 noon.
	103·8 6 p.m.		102·6 6 p.m.
27th day .	101·4 6 a.m.	37th day .	100·8 6 a.m.
	102·6 noon.		100·8 noon.
	103·6 6 p.m.		102·0 6 p.m.
28th day .	103·2 6 a.m.	38th day .	101·6 6 a.m.
	103·4 noon.		100·0 noon.
	105·0 6 p.m.		101·0 6 p.m.
29th day .	104·6 6 a.m.	39th day .	100·8 6 a.m.
	104·6 noon.		100·0 noon.
	105·8 6 p.m.		101·6 6 p.m.
30th day .	103·6 6 a.m.	40th day .	100·6 6 a.m.
	104·6 noon.		100·0 noon.
	105·0 6 p.m.		— 6 p.m.

No further fever.

CASE IV.—Filly, aged 1 year, received the following inoculations :—

29/5/05 20 cc. of heartwater blood by subcutaneous inoculation.
30/5/05 20 cc. ” ” ” ”
7/6/05 20 cc. ” ” ” ”
8/6/05 20 cc. ” ” ” ”
15/6/05 20 cc. ” ” ” ”
16/6/05 20 cc. ” ” ” ”
20/6/05 0·25 cc. virulent horse-sickness blood by subcutaneous inoculation.

The following shows the temperature record :—

1st day .	101·8 6 a.m.	4th day .	102·0 6 a.m.
	102·0 noon.		101·6 noon.
	101·8 6 p.m.		102·8 6 p.m.
2nd day .	100·2 6 a.m.	5th day .	101·2 6 a.m.
	100·8 noon.		102·0 noon.
	102·8 6 p.m.		102·2 6 p.m.
3rd day .	101·8 6 a.m.	6th day .	102·2 6 a.m.
	101·0 noon.		101·6 noon.
	102·8 6 p.m.		102·0 6 p.m.

7th day .	102·0 6 a.m.	18th day .	101·4 6 a.m.
	101·8 noon.		100·8 noon.
	102·0 6 p.m.		102·6 6 p.m.
8th day .	101·8 6 a.m.	19th day .	102·6 6 a.m.
	101·6 noon.		103·8 noon.
	102·8 6 p.m.		104·6 6 p.m.
9th day .	101·4 6 a.m.	20th day .	101·4 6 a m.
	101·6 noon.		100·6 noon.
	102·4 6 p.m.		101·6 6 p.m.
10th day .	101·8 6 a.m.	21st day .	100·4 6 a.m.
	101·4 noon.		100·4 noon.
	102·8 6 p.m.		101·4 6 p.m.
11th day .	102·4 6 a.m.	22nd day .	100·2 6 a.m.
	102·6 noon.		100·8 noon.
	104·8 6 p.m.		101·4 6 p.m.
12th day .	103·0 6 a.m.	23rd day .	100·0 6 a.m.
	102·6 noon.		100·8 noon.
	103·4 6 p.m.		103·4 6 p.m.
13th day .	101·4 6 a.m.	24th day .	104·0 6 a.m.
	100·6 noon.		104·8 noon.
	101·6 6 p.m.		105·8 6 p.m.
14th day .	101·2 6 a.m.	25th day .	103·0 6 a.m.
	100·4 noon.		103·8 noon.
	101·6 6 p.m.		103·8 6 p.m.
15th day .	101·2 6 a.m.	26th day .	101·4 6 a.m.
	101·0 noon.		102·0 noon.
	101·4 6 p.m.		102·0 6 p.m.
16th day .	100·0 6 a.m.	27th day .	101·4 6 a.m.
	102·0 noon.		101·0 noon.
	102·6 6 p.m.		101·4 6 p.m.
17th day .	101·8 6 a.m.	28th day .	101·0 6 a.m.
	102·6 noon.		100·4 noon.
	103·8 6 p.m.		100·4 6 p.m.

No further fever.

CASE V.—Foal, aged six months, received the following inoculations.—

29/5/05	20 cc. of heartwater blood by subcutaneous inoculation.
30/5/05	20 cc. „ „ „ „
7/6/05	20 cc. „ „ „ „
8/6/05	20 cc. „ „ „ „
15/6/05	20 cc. „ „ „ „
16/6/05	20 cc. „ „ „ „
20/6/05	0·25 cc. of virulent horse-sickness blood by subcutaneous inoculation.

The following is the temperature record :—

1st day .	101·6 6 a.m.	3rd day .	101·2 6 a.m.
	101·0 noon.		101·3 noon.
	101·4 6 p.m.		102·8 6 p.m.
2nd day .	101·6 6 a.m.	4th day .	101·0 6 a.m.
	101·4 noon.		101·4 noon.
	103·0 6 p.m.		102·6 6 p.m.

5th day .	101·6 6 a.m.	17th day .	101·6 6 a.m.
	101·6 noon.		101·0 noon.
	102·8 6 p.m.		102.4 6 p.m.
6th day .	102·0 6 a.m.	18th day .	101·4 6 a.m.
	101·6 noon.		101·2 noon.
	101·6 6 p.m.		102·4 6 p.m.
7th day .	101·0 6 a.m.	19th day .	103·0 6 a.m.
	102·2 noon.		103·6 noon.
	102·6 6 p.m.		104·0 6 p.m.
8th day .	101·4 6 a.m.	20th day .	101·6 6 a.m.
	101·4 noon.		101·2 noon.
	102·0 6 p.m.		102·4 6 p.m.
9th day .	101·0 6 a.m.	21st day .	101·8 6 a.m.
	102·2 noon.		101·0 noon.
	102·8 6 p.m.		101·8 6 p.m.
10th day .	102·0 6 a.m.	22nd day .	101·8 6 a.m.
	101·6 noon.		101·2 noon.
	102·8 6 p.m.		102·8 6 p.m.
11th day .	102·6 6 a.m.	23rd day .	101·8 6 a.m.
	103·0 noon.		101·0 noon.
	104·0 6 p.m.		103·0 6 p.m.
12th day .	103·0 6 a.m.	24th day .	102·2 6 a.m.
	102·0 noon.		103·2 noon.
	102·0 6 p.m.		103·0 6 p.m.
13th day .	101·2 6 a.m.	25th day .	103·0 6 a.m.
	101·2 noon.		101·0 noon.
	101·6 6 p.m.		101·4 6 p.m.
14th day .	101·4 6 a.m.	26th day .	101·2 6 a.m.
	101·4 noon.		101·8 noon.
	101·4 6 p.m.		102·8 6 p.m.
15th day .	101·4 6 a.m.	27th day .	101·4 6 a.m.
	101·2 noon.		102·2 noon.
	102·0 6 p.m.		102·0 6 p.m.
16th day .	101·6 6 a.m.	28th day .	101·2 6 a.m.
	101·4 noon.		102·0 noon.
	101·6 6 p.m.		102·0 6 p.m.

No further fever.

CASE VI.—This is the animal A already reported by Dr Edington, full details of which will be found in this *Journal*, Vol. XVII., Pt. 2, June 1904.

The animal received two inoculations of 20 cc. heartwater blood on successive days under the skin; there was a sharp reaction and the animal died on the twelfth day of the disease.

CASE VII.—This is Dr Edington's animal B, which also received two inoculations; it had a moderate reaction lasting about ten days, and it then recovered.

We have here then a record of seven cases in which horses have been inoculated with heartwater, and it may be noted that Cases VI. and VII. are the only cases in which definite fever was produced by two inoculations; in all the other cases more numerous inoculations were given. Cases II., VI., and VII. are peculiar also in another aspect. In all three cases blood was taken and preserved, and in every case found to produce horse-sickness after periods varying from one month to twelve.

The four other cases did not have this conclusive test applied, the evidence in their case being sought in the presence or absence of protection against virulent horse-sickness blood; I may mention here that Cases IV. and V. were tested with the same blood, and in equal doses, as gave a reaction in Case III. If we examine these four cases we find that in Case I. a large dose of virulent blood was given, viz., 5 cc.; this dose is quite sufficient to overcome any small amount of resistance and protection, yet even with this dose the animal recovered. In Case III. we have a somewhat similar result, only in that case the reaction was slighter.

In Cases IV. and V. there does not appear to be any reaction to the virulent blood. In all four cases, therefore, we find evidence of some protection against virulent horse-sickness, although in some of them the heartwater inoculations produced little fever.

Finally, I would point out that Case VII. (Edington's B) answered to both tests, and has since been inoculated with 10 cc. of virulent horse-sickness blood, without fever. From this record of seven cases it may be observed:—

First.—That horses can be infected with heartwater, although with some difficulty.

Second.—The infection thus produced is like horse-sickness, both in the *post-mortem* conditions found and in the fact that the blood will preserve and produce the disease in a clean horse after many months.

Third.—That animals which recover from an attack of fever produced by inoculation with heartwater have received some protection against horse-sickness.

Apart from the academic interest of these cases, there is raised a practical question of great importance. Can the inoculation of heartwater blood into horses be used practically to give them protection against horse-sickness?

The answer, I think, must be given in the affirmative, although at the same time it may be recognised that it is far from being an ideal vaccine.

Its two greatest faults are obvious: *First*, There is a fairly high mortality, 28·5 per cent., and *Second*, The vaccine cannot be preserved. Thus all the inoculations must be made at the centre or centres where it is prepared.

There are several minor faults, such as the necessity of making several inoculations and the expense incurred by keeping a strain of heartwater going.

These difficulties limit the use of this vaccine to places where heartwater and horse-sickness are both endemic, and to those Institutes where a strain of heartwater is maintained, in a horse-sickness area.

Thus in the eastern districts of the Transvaal, where both diseases are endemic, the farmers and veterinary surgeons might well use the blood of animals sick with heartwater in this way. In these cases numerous inoculations should be given, as the heartwater blood is likely to be of low virulence and the mortality due to inoculation extremely low. These inoculations would not injure the horse in any way, and there is no need to stable the horse unless a definite temperature reaction is observed. But the best use of this method could be made in a large centre such as Pretoria, where a strain of heartwater is maintained for scientific purposes. The method I should recommend for adoption there would be as follows:—

First Inoculation.—20 cc. of fresh heartwater blood to be given by subcutaneous injection (this blood should not have been drawn from the goat for a longer time than four hours).

Second Inoculation.—The same dose should be repeated on the following day, and fresh blood used if obtainable; if not, blood kept in an ice chest over night must be given.

Third Inoculation.—The same dose of fresh blood should be given eleven days after the first inoculation.

Fourth Inoculation.—The same dose of fresh blood should be given (or if not obtainable, the preserved) as in second inoculation. Further inoculations could be given with advantage if no reaction has been obtained by these; but, apart from any marked reaction, these animals will have gained sufficient protection to enable them to pass safely through a horse-sickness season, or, if any of them do contract the disease, to ensure recovery in the majority of cases.

WHITE SCOUR IN CALVES AND ITS PREVENTION BY MEANS OF SERUM INJECTIONS.*

By C. O. JENSEN, Professor at the Royal Veterinary and Agricultural College, Copenhagen.

THE occurrence of white scour in a particularly persistent and malignant form on a farm induced me to begin the bacteriological investigations with regard to the etiology of the disease, the results of which I published [1], [2] in 1892, and which have since then been in various ways completed. The extremely heavy mortality among the calves in this valuable stock further induced me, as soon as I had concluded the bacteriological researches necessary to elucidate the cause of the disease, to take in hand experiments bearing on its prevention. After the usual methods of treatment (disinfection, isolation, cleansing, etc.), although carried out with special care, had led to only a slight and temporary improvement in the state of affairs, two sets of experiments were instituted on the farm, the object of them being to immunise the cows far advanced in pregnancy in the hope that the embryos would also thereby be immunised. A number of the cows received at regular intervals subcutaneous injections of large quantities of filtered white-scour cultures, while others were similarly treated with small quantities of living bouillon cultures. The results of these experiments were entirely negative. Possibly this was ascribable to the circumstance that the animals received much too small a quantity of the culture, or it may be that it is quite impossible to produce the desired effect on the fœtus in this way.

At the same time experiments of different kinds were begun in the laboratory. It was found to be impossible to prevent infection by previous or simultaneous treatment of the calves with antiseptic agents; indeed, the experiments in this direction yielded surprising results, for they showed that creolin water, solutions of pyoctanin, and other comparatively harmless substances set up in new-born calves a severe and generally fatal enteritis, which was mainly due to an invasion of colon bacilli through the damaged mucous membrane.

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* Translated from the "Zeitschrift für Thiermedicin," Vol. IX., 1905, p. 321-399.

A symptomatic medicinal treatment also proved of no value. Inasmuch as my observations with regard to the spontaneous occurrence of the disease appeared to me to indicate that its onset might possibly be connected with a delayed commencement of secretory activity on the part of the digestive glands, a series of laboratory experiments was undertaken, partly with isolated ferments, partly with extracts of gastric and intestinal mucous membrane and pancreas added to the milk; but all had the same result, viz., that the infection was not prevented, and that the disease ran an unimpeded ordinary course.

A change of diet was equally unsuccessful; it is true that feeding with a decoction of barley or oatmeal, as a substitute for milk, was tolerated by the young calf, but it exercised no influence on the admission of the infective material into the intestine or its multiplication there. Boiling of the milk was found to be specially unfavourable; for on the before-mentioned infected farm and two others it caused a decided increase in the death-rate. Laboratory experiments showed that new-born calves for the first twenty-four hours after birth cannot stand boiled or pasteurised milk; when thus fed they almost invariably die with the symptoms of a severe (often hæmorrhagic) enteritis. Bacteriological examination showed that we here had to do with a condition similar to that produced by the administration of the above-mentioned antiseptics, the enteritis being ascribable to an invasion by colon bacilli, which after death were found In great numbers in the blood, and were proved by experiments on other calves to be capable of producing the disease.

It was about this time that the antitoxic sera were discovered. Since it is well known that it is only for a short time after birth that calves are susceptible to white scour, there must in this disease, more than in any other, be room for serum-treatment, provided it is at all possible to prepare a serum capable of immunising against it. By treating horses, partly with dead and partly with live cultures, it was found possible, but certainly with some difficulty, to prepare a serum which was proved by experiments on guinea-pigs to be in a high degree bactericidal, and which laboratory experiments also showed to be capable of preventing with certainty the otherwise invariably fatal effect of artificial infection.

In 1895-96 and 1896-97 experiments were begun with this serum on the before-mentioned farm, where an assistant took up his temporary abode, in order that he might be in a position to treat the new-born calves immediately. About half of the calves were left untreated, so as to serve as controls. The results were as follows:—

1895-96. Of thirty calves treated with serum 57 per cent. died. Of thirty-two control calves 75 per cent. died.

1896-97. Of forty-three calves treated with serum 46·5 per cent. died. Of sixty-three control calves 76·2 per cent. died.

The result was thus not satisfactory.

Since experiments on guinea-pigs showed that my serum was not equally protective against all strains of white-scour bacteria, and was, indeed, quite inactive against some of them, I decided to submit the entire group of organisms to which the white-scour bacteria belong (the typhoid-coli-swine-fever group) to a thorough investigation. These researches, which are practically completed, and of which only a fraction has previously been published,[4], [5], [6] were of a very extensive character, since many hundreds of strains were examined. The investigations embraced, *inter alia*, the power of the bacteria to ferment or split up a large number of the sugars, polyvalent alcohols, and organic acids, as well as their capacity for using albumen, albumose, peptone, amid-combinations, ammonia, etc., as sources of nitrogen.

By means of these rather laborious investigations I was able to arrive at a better understanding of the group, to recognise in it distinct sub-groups, and in a reliable way to distinguish from one another a number of forms which by the methods ordinarily employed would have appeared completely identical. I may say that I was thus able to distinguish three constant varieties of the typhoid bacillus, and that I was also able to recognise great differences between the various para-typhoid bacilli and the strains of swine-fever bacilli.

Continued serum experiments further showed that the various white-scour cultures exhibited great differences with regard to their agglutinin and bacteriolysin. In a general way it may be said that a serum prepared by using a single strain of culture is active against that and some other strains, both as regards agglutination and protective effect (guinea-pig and calf experiments), but is inactive against most of the cultures isolated from other farms.[8] Indeed, from the same farm one may isolate cultures of which one is influenced by a serum while the other is not, and also cultures which differ with respect to biological characters. However, such complex conditions are not frequent, but, since they may not be devoid of importance with regard to the practical application of a serum, I shall return to the subject later.

If the serum treatment was to be made available for more than a single farm, one had either to prepare a series of different white-scour sera, or one had to attempt to immunise the same horse with as many different strains as possible; in other words, to prepare what subsequently came to be known as a "polyvalent" serum. This caused no particularly great difficulty, although the matter was not concluded by using as many cultures as possible. During recent years I have worked with sera of varying "valency," especially with a serum which was obtained by using a dozen specially selected strains, and which has given satisfactory results on about 70 per cent. of the infected farms. I consider it impossible to prepare a single serum which will be appropriate for all outbreaks of the disease, partly because, as I shall show later, there are enzoötics with bacteriological circumstances quite different from the ordinary, and partly because the number of different forms of the colon bacillus is certainly very great. On the other hand, I regard it as highly probable that, by a continued selection of cultures, and by using a large number of these, one may be able to prepare a serum which will be suitable for 90 per cent. of the outbreaks. The majority of the remaining outbreaks can certainly be treated successfully with another serum (paracoli serum); for most of these are caused by the so-called paracoli bacilli, by means of which it is also possible, though more difficult, to prepare a useful immunising serum. In my opinion, it will be very difficult, and scarcely practicable, to prepare a single serum appropriate both for the coli strain and for the paracoli forms; that is to say, a serum which would be in a still higher degree polyvalent.

When I continued to work with a serum of the above-mentioned valency, and did not attempt to obtain a still more active one, that was because of various circumstances; partly it was because my time was largely taken up with other work, so that it would have been inconvenient for me to enter into direct communication with too large a number of breeders, and partly because I wished to obtain light on a number of fundamental questions, which could best be answered if the available serum was active only against a number of the occurring strains. For example, the question whether the aglutinating power of a serum might be taken as a measure of its bactericidal action on individual varieties, and whether a grouping of the varieties such as had been indicated by the fermentation experiments coincided with that which would be indicated by the action of the serum. To this was added the circumstance that it seemed to me that it would be best if the State took over the manufacture of the various immunising sera, and had it carried out in a central laboratory devoted to the investigation of the more important infectious diseases occurring in the country. In Denmark the diphtheria serum is manufactured in a special State Laboratory, and at the present time negotiations are proceeding regarding the erection of a similar laboratory for the preparation of sera against animal diseases, and for the scientific investigation of the latter.

It was mainly owing to these different circumstances that I did not carry into practice to a larger extent the results of the laboratory experiments.

Since the etiological circumstances of the disease are intimately connected with the preparation of the serum and serum treatment, I may be permitted to deal shortly with the principal facts, at the same time referring the reader to a more exhaustive account of them which I have written for Vol. III. of Kolle and Wassermann's *Handbuch der Pathogenen Microorganismen.*

In my first report I recognised the great resemblance between the white scour bacterium, and the, at that time, little known bacillus coli. In a subsequent report I identified these two bacteria, since I regarded the white scour bacterium as a more virulent race of the ordinary bacillus coli. This view was confirmed by the investigations of Willerding,[15] Schütz,[16] and others. In a report published in 1900[8] I recognised that the bacteria from different outbreaks of the disease were not identical with one another, and that white scour, even in infection experiments, may be caused by a series of more or less easily recognised varieties of the bacillus coli. In 1899 appeared the large and very interesting work by Poels,[14] a work which brought new light on several points; for example, it was there shown that some outbreaks of white scour are caused by an infection with paracoli bacilli, and that in rare cases the bacillus pyocyaneus may also be responsible for the occurrence of the disease. Poels further confirmed the correctness of my assertion that the bacillus coli is by far the most frequent and important of the white scour microbes; on the other hand, he disagreed with my view in so far as he assumed that the white scour bacterium is not identical with the bacillus coli of the normal bowel, but a nearly related virulent form. Lastly, Poels showed that the bacillus coli is able to penetrate the body by means of the umbilical vessels, and that there occur cases of the disease in which there has undoubtedly been a simultaneous infection by the mouth and through the navel with the same or with different varieties of bacteria (for example, bacillus coli and streptococci). My own further investigations[7] have confirmed the occurrence of paracoli enzootics, and have also shown that the bacillus ærogenes and proteus forms are rarely-occurring white scour bacteria. Furthermore, I have had the opportunity to observe cases where there had undoubtedly been a pure coli infection through the umbilical vessels (septicæmia), others in which there had been a combination of intestinal and umbilical infection with coli and paracoli bacilli, and still others in which there had been a mixed infection. Such cases, however, were exceptional, and among the numerous cases examined it was nearly always found that the infection was purely intestinal, in other words, proper white scour.

In opposition to Poels, I consider the coli forms occurring in white scour as identical with the intestinal bacillus coli forms. In support of this view I may point out that it is not a single, but numerous varieties or races of the bacillus coli which can cause the disease, and also the fact that in the case of most new-born calves feeding with boiled milk, even on entirely healthy farms, or under the best of laboratory conditions, sets up a severe fatal coli enteritis, and that the same effect can be produced by a dose of creolin water or similar antiseptic.

The works of Bongert[17] and Jœst[18] have confirmed the view that the colon bacilli must be regarded as the cause of the immense majority of outbreaks of white scour. On the other hand, French investigators have arrived at quite a contrary opinion. In an article regarding the "diarrhœa of young calves," Lesage and Delmer[11] deny the etiological importance of colon bacilli, and assert that the disease is not produced by infection through the mouth, but, on the contrary, by the penetration of microbes through the umbilical vessels. Lastly, in their opinion, the cause of the disease is a pasteurella. Similar results were obtained by Nocard[10] during his investigations regarding white scour in Ireland, and Nocard's views were supported by Mettam[12] (as the result of his own investigations?). In the above-mentioned Handbuch I have criticised at length the results obtained by the French investigators, and here, therefore, I need only point out the following facts. The pasteurella bacterium which was discovered by them was incapable of infecting by the alimentary canal, even in very large doses, while the effect produced by subcutaneous inoculation was inconstant, and even intravenous inoculation did not always produce the disease. Pasteurella microbes, as is well known, occur in the mouths of healthy calves, and, as my investigations have shown, they may also sometimes be recognised in the intestinal contents of such calves. My own experiments formerly carried out have proved that the disease may be communicated to healthy milk-fed calves by giving them *per os* a minimal quantity of fæces of a diseased calf, and through very numerous experiments carried out by myself and Poels it was ascertained that the same effect is produced by pure cultures. In many cases a deadly infection was produced by feeding with only

4. The disease caused by the bacillus pyocyaneus, which has hitherto been observed only by Poels, has profuse diarrhœa and increasing weakness for its chief symptoms, and, *post-mortem*, it shows red specks on the intestinal mucous membrane and degeneration of the liver, but no swelling of the spleen. The bacillus pyocyaneus occurs in almost pure culture in the intestine, while the blood, on the contrary, is sterile.

5. The proteus infection sets in when the calf is a few days or perhaps a week old, and it runs a slow course. The fæces are stinking, but never mixed with blood. At the *post-mortem* one finds the intestines strongly distended with gas, the serous and mucous membranes pale, often to a pronounced degree, and the latter after death rapidly assume a macerated appearance. The spleen is not swollen; some of the mesenteric glands are enlarged, but only slightly hyperæmic. Proteus bacteria are found in considerable numbers in the intestinal contents, (which in normal calves contain no bacteria that liquefy gelatine); on the other hand, the spleen and the mesenteric glands are free from bacteria. Quite the same clinical and pathological picture can be experimentally produced by infection with isolated proteus bacteria. The proteus infection has also been observed by Raebiger and Reimers.[19]

As already stated, I have not myself had any experience with the pyocyaneus infections. With some proteus forms obtained from cases of white scour I have by inoculating a goat produced a serum possessed of strong agglutinating properties; hitherto I have carried out practical experiments with it to only a slight extent, and they have not as yet yielded any useful result. An ærogenes serum was obtained by means of subcutaneous and intravenous injections into horses, and this had strong agglutinating and bactericidal properties, and in laboratory experiments with calves it yielded satisfactory results. Owing to the rare occurrence of the disease caused by ærogenes bacilli, no attempt has yet been made to combat it by serum injections. So far as I can see, the preparation of an ærogenes serum presents no difficulty, and the conditions would certainly be exactly the same as those necessary for the preparation and use of the coli serum. Experiments were undertaken with monovalent, divalent, and polyvalent coli serum; with paracoli serum; and with serum which immunised both against different coli and paracoli infections.

COLI SERUM.

For the preparation of serviceable coli immunising serum the horse is certainly the most appropriate. This animal stands the necessary operations comparatively well, and after proper treatment furnishes a useful serum. To this has to be added the well-known fact that the blood of the horse yields a comparatively large quantity of clear serum, which is only in a very slight degree toxic. According to my experiments, the ox appears to be unsuitable for the preparation of the serum, although after long-continued treatment one succeeds in imparting bactericidal properties to the serum of this animal. The goat appears to be a more suitable animal. According to Bongert's experiments, very good results are obtained with the sheep, but owing to the small amount of blood obtainable this animal is not convenient for the preparation of serum in large amount. This applies with still greater force to rabbits, although these may be immunised and furnish an active serum (Jœst); during the treatment, however, these animals are apt to succumb from a progressive cachexia.

According to my experiments, which, however, have not been very extensive, the different breeds of horses do not in this respect show any great difference. I obtained serviceable serum from light as well as from heavy horses, and I did not observe any great difference between young and old horses.

As regards the method of inoculation, I hold the intravenous injection to be by far the best. By feeding with a very large quantity of culture it is possible to confer on the serum some agglutinating power, though certainly not very strong, but it is doubtful whether any bactericidal action can be produced in this way. Subcutaneous injections are followed by rather large and important inflammatory infiltrations, which often end with extensive necrosis of the connective tissue, muscular tissue, and skin. Even by proceeding with the greatest care one cannot always prevent this unfortunate occurrence, which may very easily cause a serious delay in the immunising process. It would appear that the various cultures behave somewhat differently as regards their effect, for there are some which very easily cause necrosis, while others have little tendency to do this. This untoward result may certainly be to a large extent prevented by trituration of the bacilli before the inoculation; but it cannot be altogether averted in this way. By the use of subcutaneous injections one obtains a comparatively active serum, although, both with respect to its agglutinating power and bactericidal properties, it falls far behind the serum which may, within the same period, be obtained by means of intravenous injections. For example, in one series of experiments three horses were treated with a particular culture: one by feeding, a second by subcutaneous inoculation of triturated bacilli, and a third by intravenous injections of living bacilli; after long-continued treatment one found the following figures for their respective agglutinating power (precipitation of bouillon cultures): 1 in 500, 1 in 10,000, and 1 in 100,000. In the case of the sheep, Bongert recommends intraperitoneal injections as being generally well borne.

As a rule, I used for the inoculations bouillon cultures which had been incubated for twenty-four hours at 37°; suspensions of agar cultures appear to me to have no special advantage. As mentioned above, I found that triturated bacilli are capable of leading to the formation of bacteriolysins in the horse, and the same holds good for bouillon cultures which have been killed by chloroform or toluol. However, the use of cultures which have been killed in this way offers hardly any advantage, for the toxic effect thereby produced is not nearly so strong as when living cultures are employed. Whether cultures which have been dried and then destroyed by heat (Löffler's method) might be successfully employed, I am not able to say, as the experiments bearing on that point have not yet been completed.

As a rule, intravenous injections are quickly followed by restlessness, which may go on to attacks of colic, and also to a slight degree of fever (elevation of the temperature above 2-3°), which disappears after a period of from six to twelve hours. Absence of appetite often persists for some days afterwards. Provided none of the culture has escaped into the connective tissue, there are no local alterations; but when such an accident happens during the injection an inflammatory infiltration appears, and may end in necrosis. The injections are made with intervals of ten to twelve days, and never earlier than from eight to ten days after the disappearance of every trace of reaction to the preceding inoculation.

Not all cultures are equally good for the preparation of serum. There are strains of coli bacilli which are scarcely employable, because they do not stimulate the animal in any appreciable degree to the production of lysin. On the other hand, there are single strains which are specially appropriate for this purpose, since by their use one obtains a serum which not only exerts a particularly strong effect on the strain employed, but is also active on a larger number of coli strains than is usually the case with a monovalent serum. What is the explanation of this cannot at present be decided. The cultures in question do not possess specially pronounced toxic properties, and there is therefore no ground for supposing that such a serum ought to possess decided anti-toxic as well as bacteriolytic properties.

Whereas the usefulness of different coli strains is thus without doubt variable, the greater or slighter virulence of particular cultures does not appear to be of importance in this connection. Cultures which had been kept for years in sealed glass tubes were found to be serviceable as soon as they were sub-cultured; among my best cultures are some which have been, with occasional sub-culturing, kept in the laboratory for a series of years, and this without their having been inoculated into animals in the interval. Whether a culture which has been found to be not of much use for serum production might be improved by passage through one or several sucking calves, I am unable to say, but it does not appear to me that such is the case.

It is not possible to lay down a general rule with regard to the most appropriate dose; for the different strains, as is sometimes shown by guinea-pig experiments, for example, may exhibit pretty considerable differences in toxicity. Ordinarily I injected on the first occasion a small dose, commonly 0·25 to 0·50 cc. of a twenty-four hours' bouillon culture; if this provoked a prominent reaction, the same or a somewhat similar dose was used after about fourteen days. If the first injection was well borne by the animal, the dose was doubled on the next occasion; and at each subsequent injection the quantity was, sometimes slowly, sometimes quickly, increased, until from 10 to 20 cc. was injected at a time. As a rule, this immunising is effected without any accident, provided always that the before-mentioned precautions are observed. It may, however, happen that marked intoxication symptoms ensue in a horse; progressive emaciation, anæmia, and weakness ought to be specially mentioned. These may either cause the death of the animal or necessitate its destruction, or after many months of continued careful treatment they may gradually disappear. During this illness the immunising power of the serum appears to be entirely lost, but when the animal recovers, and fresh inoculations are practised, it is re-acquired. In combination with such emaciation, or more rarely as an independent morbid condition, a chronic inflammation of the tendon sheaths may set in, with a tendency to relapse and to extension from one tendon sheath to another. This synovitis, which is known to occur in the course of other immunisations, may last for months, causing the animal to become completely crippled, and necessitating its destruction. Treatment with serum, taken from another horse, or from the diseased one at an earlier period, appears to exert no curative effects on this nasty complication, which may set in unexpectedly and cause the death of a previously excellent serum horse.

The preparation of a monovolent serum is not attended with any great difficulty, except that, as already mentioned, some cultures are not at all appropriate for immunising. The monovalent serum is serviceable against the strain of bacilli used in its preparation, and at the same time also against a series of other strains; indeed, it is sometimes serviceable against strains which in some point or another exhibit striking differences from the one employed in immunisation. Bongert advises that when white scour breaks out on a farm a culture ought to be obtained from one of the diseased or dead calves, and that this should be used in the form of intraperitoneal inoculations to immunise a sheep. After the course of four or five weeks the sheep ought to be so strongly immunised that its serum will suffice to protect new-born calves in doses of from 20 to 30 cc. Undoubtedly in many cases this or a similar procedure will prove suitable; although, ordinarily, several months elapse before the serum animal reaches the desired degree of immunity. In practice, however, the result does not by any means always come up to expectations, especially when one has to deal with a large farm on which the disease has been prevalent continuously for several years. In such a case the serum treatment will often save only the half, or a little more, of the new-born calves, and in the diseased and dead calves one will then find coli forms which are not influenced by the serum, and which can often, by their different fermentative power, be with certainty distinguished from the strain of bacilli originally isolated and employed for the preparation of the serum. Indeed, it is not at all rare to find virulent coli forms and proteus forms simultaneously present on the same farm.

I had an opportunity to follow a particularly fine example of this for a couple of years. On a very large farm, where white scour had existed for many years, the disease one year occurred in a rather malignant form. A number of calves were examined, with the result that numbers of coli bacilli were found in the spleen, blood, and glands; whereas plate cultures, made with intestinal contents, showed numerous coli colonies and a few liquefying forms. A serum which in laboratory experiments had proved itself active against the isolated coli form was brought into use at the farm in question, with the following results. The control calves, just as before, died one or two days after birth, while those treated with serum died after the lapse of about a week. Whereas the former showed severe enteritis, swelling of the spleen, etc., and had masses of coli bacilli in their blood, the calves which had been treated with coli serum showed what has been previously described as the characteristic picture of the proteus infection (pale, gas-distended intestine, no swelling of the spleen), and even twenty-four hours after death no coli bacilli were present in the blood or organs of the carcase, but when the intestinal contents were used for inoculating media the resulting colonies were almost exclusively liquefying ones. The isolated proteus cultures, when fed to sucking calves, were found to cause exactly the same type of disease as had been observed spontaneously in the serum-treated calves. The explanation of these circumstances, which I observed in a large number of animals in two successive winters, is extremely simple. The treatment with coli serum conferred immunity against the highly virulent coli bacilli, and thus made it possible for the less virulent proteus bacilli to exert their slower effects.

The observations here referred to show that one is not able by means of a monovalent serum to combat the disease in all cases, nor even always on a single farm. Our efforts ought therefore undoubtedly to be directed towards obtaining a polyvalent serum of the highest possible valency; but, as the cases just cited show, we must be prepared to find that in practice even such a serum may not give satisfactory results in every case.

There are certain difficulties in the way of obtaining a polyvalent serum. When we inject a horse with a mixture of several bacteria, we have no assurance that the whole of the bacteria will exert on the horse such an immunising influence that its serum will possess protective properties against each one of the forms employed. For example, I submitted one horse to repeated intravenous injections of a mixture of bouillon cultures of one paracoli bacillus and ten different coli strains. After a period of continuous treatment the blood was found to contain anti-bodies against the paracoli bacillus. The intravenous injections were continued with the omission of the paracoli bacillus, and now anti-bodies were formed against a single one of the coli forms, but none against the others. When, during the later inoculation, this particular coli strain was omitted from the culture mixture, the blood gradually acquired the power of influencing most of the coli strains used. But even after a period of more than a year there were some of the cultures which did not appear to be influenced by this otherwise very active serum.

In the preparation of serum, therefore, it is uudoubtedly important to carefully select the cultures, and, by frequently repeated examination of the behaviour of the serum towards each of the forms used, to strike out those which have a less favourable influence, and also those which, during the immunising process either exercise a "dominating" influence or behave in a passive way.

In order to obtain a polyvalent serum I likewise employ intravenous injections, with intervals of about fourteen days. Each strain is cultivated separately in bouillon at 37°, and after twenty-four hours the desired quantity of each separate culture is measured, and the mixed bouillon cultures are injected. The cultivation of the whole of the strains employed in the same bouillon is not advisable; for one never knows how many or what strains succeed in multiplying at all in the culture. There appears to be no objection to beginning the immunisation with single strains, and gradually increasing the number of these later.

PARACOLI SERUM.

The varieties of bacteria belonging to this group are characterised in general by possessing much stronger pathogenic properties than is the rule with the coli forms. Even in calves this is seen quite distinctly. Not only have the cases of white scour caused by the paracoli bacilla an acute and decidedly septic character, but even in older, or it may be half-grown calves, both sporadic cases and enzootics of paracoli enteritis occur. Owing to this greater virulence inoculations into horses must be undertaken with great care, for an intravenous injection of 0·1-0·25 cc. of a bouillon culture may, in the case of a horse which has not been previously treated with this form, determine a severe, acute, dangerous illness; and even later, during the process of immunisation, intoxication and inflammation of the tendon sheaths is more apt to occur than in horses used for the preparation of coli serum. If one is already in possession of a paracoli serum, one should give a preliminary serum injection, and follow that up by the first injection of culture. This, however, is not the only difficulty which lies in the way of obtaining a paracoli serum; many, and perhaps even the majority of strains, appear to be not at all appropriate for serum production. It is true that they determine the formation of an abundant amount of agglutinin, but, on the other hand, little or no formation of immunising bacteriolysin. This is quite in agreement with experience in the preparation of swine fever serum, and that is not astonishing, in view of the near relationship between the paracoli bacilli and the swine fever bacteria.

I was fortunate enough to come into possession of what proved to be a useful strain. The serum obtained with it proved serviceable in experiments on calves, and the results obtained with it on some farms were satisfactory. Even in some outbreaks of paracoli enteritis among older calves, the serum proved capable of preventing the disease.

The agglutinating power of a paracoli serum is usually very considerable, and it is remarkable that it is present in only slightly diminished amount in the blood of horses even a year or more after the last injection. The blood of horses immunised against coli bacilli also retains its agglutinating properties for a long time, but this appears gradually to become weaker than is usually the case with paracoli agglutinin. Whether the bacteriolysins are to a similar extent retained in the blood after the regular inoculations have been stopped, I cannot at present say. A paracoli serum is never active against a coli form, just as a coli serum is never active against paracoli bacilli.

I have not made any attempt to prepare a polyvalent paracoli serum. For the reasons mentioned above, its preparation would certainly be attended with a good many difficulties, and this applies with still greater force to the preparation of a polyvalent serum for use against both the paracoli and the coli forms. On the other hand, no great difficulty was encountered in obtaining by the use of two specially selected strains, a serum very active against a paracoli form and a single coli form.

As numerous intra-peritoneal inoculations into guinea pigs have shown, the action of a serum prepared in the above described way is a bacteriolytic one. Whether it has at the same time an anti-toxic action I do not know with certainty. As is well known, it is not possible to detect powerful toxins in the cultures of most coli forms; and, in spite of numerous experiments, varied in many ways (cultivation in albuminous solutions, blood, intestinal contents, etc.), I did not succeed in inducing the white scour bacteria to produce any free toxin worth mentioning.

The estimation of the activity of coli serum against different coli forms presents some rather important difficulties. It is true that for this purpose one may use new-born calves, but this is rather costly, and when they are obtained from dealers' premises they have very often been infected during or immediately after birth, either through the umbilical region or the alimentary canal, and, in consequence of this, experiments carried out on them are very unreliable. Here, in Copenhagen, it is extremely difficult to obtain the necessary healthy experimental calves. For this purpose the smaller experimental animals, such as rabbits and mice, are unsuitable, owing to the uncertainty with which a deadly disease is set up by the injection. Formerly, I used for the purpose guinea-pigs, which succumb to an intraperitoneal inoculation, but the different freshly isolated cultures exhibited very varying degrees of virulence when tested in this way. The lethal dose of a twenty-four hours' bouillon culture may vary between 0·2 and 1 cc., and to that has to be added the fact that under prolonged cultivation in artificial media the cultures suffer loss of virulence. Moreover, different guinea-pigs of the same weight and age react very differently to the same dose of the same culture; one may die of an acute peritonitis, while another remains unaffected. On these grounds it is obvious that it is very difficult to employ the peritoneum of the guinea-pig as a reliable criterion. It would, of course, occur to one to transform the culture in such a way as to make its pathogenic properties for guinea-pigs stronger, and therefore more uniform. By inoculating into the peritoneum, then sowing out the heart-blood of the dead guinea-pig in bouillon, next day inoculating a fresh guinea-pig intraperitoneally, cultivating from the heart-blood of the latter, and so on, one sometimes succeeds in intensifying the virulence to a very considerable degree. Two different cultures, which, when freshly isolated, killed guinea-pigs (of 500 gr. weight) in sixteen to twenty-four hours with a dose of 0·5 cc., by alternating inoculation and cultivation in bouillon had their virulence so increased that a dose of 0·01 cc. was certainly fatal to guinea-pigs of the above-mentioned size in about sixteen hours. By means of this culture the strength of the serum was easily determined (0·001 cc. protected 500 gr. guinea-pigs against the certainly fatal dose). This, however, is scarcely a practical method. In the first place, it requires a large number of guinea-pigs; secondly, it is by no means possible to intensify the virulence of all coli cultures in the manner described; and, lastly, it is very doubtful whether those figures which one obtains as the measure of the activity of the serum against an artificially cultivated, partially virulent culture ought to be allowed to have the same value when the question is that of the virulence of the "wild-growing" coli forms for calves. Possibly one may be able to arrive at a reliable method of estimation by relying upon, not the death of the animal after the intraperitoneal inoculation, but the occurrence or non-occurrence of an infiltration after subcutaneous inoculation. Meanwhile such a method is not available, and it is therefore possible that the dose of the serum which I have hitherto used for calves is much greater than is actually necessary.

We are also without a method for estimating the strength of the paracoli serum. However, owing to the fact that a number of small rodents, especially *arvicola arvalis* and *arvicola agrestis*, are usually very susceptible to infection with bacteria of this type, it is probable that these animals may be successfully employed for the purpose, either by feeding them with culture, or inoculating them intra-peritoneally with it.

As is well known, the agglutinating and the bacteriolytic powers of a serum do not exactly coincide; the former may be strongly developed while the latter is completely absent. It is possible that this is the case with regard to white scour, but upon the whole it may be said that if a freshly-isolated culture is agglutinated (precipitated) by a diluted serum (1 in 1000 to 5000) the latter will almost without exception show itself bacteriolytic with regard to the bacteria in question; and conversely. In other words, the serum will be available for use on the farm from which the culture was derived. In the absence of accurate methods of estimation, I have made somewhat extensive use of this agglutination method, and, although it is not absolutely reliable, it has nevertheless proved of no slight service.

As an addition to the serum, I have generally used chloroform; but dilution with carbolised water, or the addition of carbolic acid, such as is commonly practised with other kinds of serum, does not interfere with the activity of this serum. When kept in the dark and in a cool place, both the coli and the paracoli serum retain their properties for a long time.

The way in which I have hitherto employed the serum on infected farms was as follows. Agar plates were prepared from the spleen or the mesenteric glands, and at the same time gelatine plates from the intestinal contents, in order to detect any proteus infection that might be present there. On the agar plate cultures five or six colonies were examined; if no colonies developed on these plates, a number of similar coli colonies from the intestinal plates were used. The isolated cultures were examined with regard to their behaviour towards lactose bouillon in fermentation flasks, by which test the paracoli infection can be recognised. At the same time, one employs the agglutination test with one or several sera, using dilutions of 1 in 50, 1 in 100, 1 in 500, 1 in 1000, or even 1 in 2000 and 1 in 3000. If the serum in a dilution of 1 in 500 to 1 in 2000 causes a precipitate, it may be taken as probable that it will prove serviceable. The serum I have hitherto sent out in measured doses of 20 cc.; as in the case of other sera, no particular care is necessary when carbolic acid has been added to it. The whole dose is administered subcutaneously at one time, and this ought to be done as soon after birth as possible. Any special precautions in making the injection are superfluous.

If our view of white scour is correct, that is to say, if infection is the chief cause, and dietetic errors only of subordinate importance (and this is hardly open to doubt), a proper serum treatment must suffice to prevent the disease, and all troublesome measures (isolation, disinfection, special diet, or care and cleanliness in feeding, etc.), are then superfluous. On that account I have prescribed absolutely no precautionary regulations on the farms where the serum treatment was carried out, and, notwithstanding that, the result has been satisfactory.

Experiments have been carried out with monovalent and polyvalent coli serum and with paracoli serum on a great number of farms in Denmark. Furthermore, under the supervision of Dr Raebiger[19]

(Halle), several very accurate and somewhat extensive experiments have been instituted in the province of Saxony. Similar experiments have also been carried out at other places in Germany with serum which I have, when requested, placed at the disposal of various colleagues. Experiments on a pretty large scale have been made in Sweden, and lastly, serum has also been sent to Norway, Finland, and Italy. Some of these experiments have already been published.

At the present moment I am not in a position to make a general statistical statement of the results obtained, partly because the whole of the information has not yet been received, and partly because it is not yet completely arranged for publication. In the meantime I shall therefore confine myself to a statement of the principal results which had been obtained two years ago.

On many farms where the disease prevailed as a malignant enzootic, the serum treatment has reduced the attacks and the fatalities to zero, even when the previous mortality had been nearly 100 per cent., and the whole of the control calves died.

On other farms the result was less satisfactory, since some deaths still occurred; but the number was notably reduced, as, for example, from 70 to 20 per cent.

On still other farms, as the laboratory experiments had led one to expect, the serum treatment had no recognisable effect.

Lastly, with regard to the use of the serum as a curative agent, it may be said that, owing to the somewhat rapid course of the disease, opportunities to gauge this seldom present themselves. However, in a series of cases I was able to observe an undeniably favourable influence on the course of the disease, the attacks being immediately cut short. At the same time, this effect is by no means constant. The paracoli serum has also repeatedly shown an unmistakable curative effect on severe enteritis in older calves.

Although the facts relating to the bacteriology of white scour are thus much more complicated than I suspected when I published the results of my first investigations in 1892, and although the prophylactic serum treatment has not yet been brought to perfection, we have every reason to expect that it will succeed in completely overcoming this scourge of cattle-breeding, and no doubt the fact that so many Institutes have now included white scour in their investigations will in an important degree contribute to this result.

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Reviews.

A Treatise on the Parasites and Parasitic Diseases of the Domesticated Animals. By L. G. Neumann, Professor in the National Veterinary School of Toulouse. Translated by George Fleming, C.B., LL.D., F.R.C.V.S. Second Edition, Revised and Edited by James Macqueen, F.R.C.V.S, Professor at the Royal Veterinary College, London. Baillière, Tindall & Cox: London, 1905.

Everyone acquainted with the merits of Professor Neumann's excellent textbook on Parasitology will admit that the late Dr Fleming did a good service to the profession in this country when he translated it into English. During the interval which has elapsed since the appearance of the first edition the work had become a little out of date, and a new edition embodying the fruits of recent researches is therefore welcome. Some of the chapters have been reconstructed, and a great deal of new matter has been introduced, while the illustrations now number 365. Detailed criticism of a work so well-known and appreciated is unnecessary. Professor Macqueen is to be congratulated on the careful manner in which he has executed his task of revision and editing. We have no hesitation in saying that no member of the veterinary profession who desires to keep his knowledge with regard to the parasitic diseases of animals up to date can afford to be without this valuable book.

Essentials of Physiology for Veterinary Students. By D. Noël Paton, M.D., B.Sc., F.R.C.P.Ed., Superintendent of the Research Laboratory of the Royal College of Physicians, Edinburgh, etc. William Green & Sons: Edinburgh and London, 1905.

As the author states in the preface, he has in this work attempted to give the essentials of general physiology and of the special physiology of the domestic animals in a form suitable to the requirements of students and practitioners of veterinary medicine. In this attempt he has been to a large extent successful, though it appears to us that the special side of the work is not quite on a level with the general. It is no doubt true that the physiology of man and that of the domestic animals have much in common, and that most of our knowledge of human physiology has been acquired from experiments upon the lower animals, but that does not lessen the importance of a knowledge of the special physiology of man for the medical student, and of the special physiology of the domesticated animals for the veterinary student. The ideal text-book of veterinary physiology, while embracing all that is important in the way of general physiology, must not omit the equally important facts of physiology which are peculiar to the various domesticated animals. As a matter of fact, Dr Paton in this work sometimes mentions special facts of human physiology as if they were general, and omits to mention at the same place other facts which are surely deserving of special notice in a text-book intended for veterinary students. For instance, in the chapter on Voice it is said that the left recurrent laryngeal nerve is apt to be pressed upon by aneurismal swellings, and that paralysis of it makes the voice hoarse or abolishes it altogether, but no mention is made of the extremely common paralysis of the nerve in horses free from aneurismal swellings, or of the important effect which such paralysis has on the respiration! As another illustration of the too human standpoint from which the facts are viewed, it may be mentioned that where anatomical descriptions occur they frequently imply that the horse and the other domesticated animals walk erect like man. In the description of articles of diet rather more space is given to the animal foods (including butter, cheese, and eggs) than to those of a vegetable nature.

In pointing out these defects in the work, we do not forget the limits which are imposed on the size of a book intended mainly for ordinary students; but we think that when a second edition is called for it will be necessary to expand considerably the parts of it which deal with the special physiology of the horse and the other animals of the farm. The present volume runs to a little over 400 pages, and includes 157 useful illustrations.

CLINICAL ARTICLES.

A REMARKABLE CASE OF DEFORMITY IN LAMB'S RIBS.

By Gerald Leighton, M.D., F.R.S.E., Interim Professor of Pathology, Royal (Dick) Veterinary College, Edinburgh.

The specimens illustrated in this article were sent to the zoological department at Edinburgh University, and handed over to me by Professor Cossar Ewart. They are of such an unique character and so difficult to account for, that it seems worth while placing them on record in the pages of this *Journal.*

All the information that could be obtained was to the effect that the ribs were taken from a lamb which had died a few hours after birth, and that a number of other lambs in the same flock were similarly affected and died. I was unable to obtain a whole carcase for dissection and observation of the other organs, hence partly, perhaps, the difficulty of being able to say exactly what is the nature of the specimen.

The appearance of the deformity will be better grasped by studying the illustrations than by a mere description. In Fig. 1 four of the ribs are shown, and it will be noticed that each exhibits upon it a swelling on the curvature, which on closer examination has the appearance of a joint. Every rib in the specimens sent was similarly deformed, including ribs from both right and left sides of the thorax.

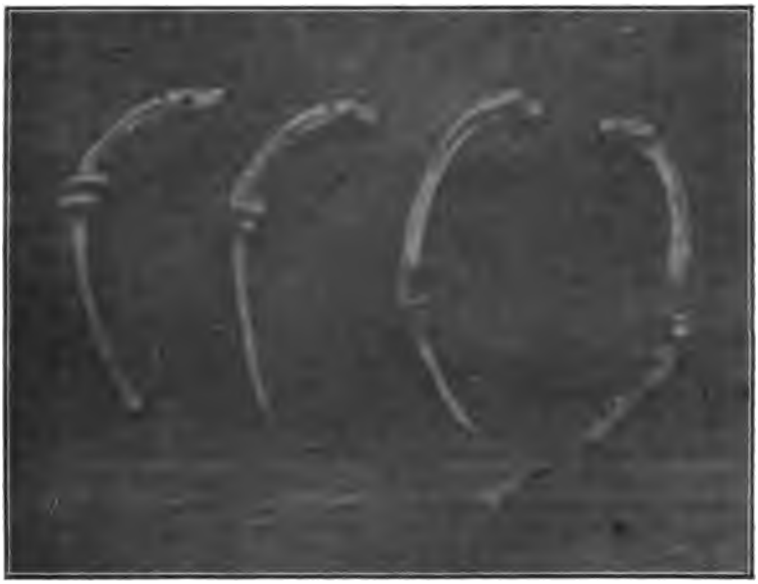

FIG. 1.

One of these joints is reproduced in an enlarged form in Fig. 2, which shows exactly the condition. It will be observed that there are no true ligaments developed, but merely some fibrous tissue on either side of what may be described as the articulating ends.

It is the etiological aspect, of course, which is of greatest interest. What was the cause or origin of the deformed ribs? Two answers only seem to be possible. The first is that the case is one of a true congenital inborn deformity, germinal in origin. The second possible explanation is that the deformity is the result of an intra-uterine traumatism. I have shown the photographs to Professor Cunningham of Edinburgh University, who is inclined to think that the case is one of intra-uterine injury leading to fracture and the formation of a false joint. He is hardly inclined to go the length of saying that it is a morphological congenital defect.

The difficulty I have in accounting for the condition in this latter way is that it is hard to imagine any injury which would produce these false joints in such a remarkably regular and bilateral manner. Every rib showed these joints and all were of the same nature. Moreover, they were on right and left ribs. Then there is the further difficulty in the statement that a number of other lambs were similarly affected. I give this simply for what it is worth, having had no means of verifying the statement, but there is no reason to doubt it. If the condition is a true germinal variation, a real congenital defect, it would most probably have cropped up again in some of the offspring of the lambs affected. But of this I have no knowledge.

The case teaches us one thing, however, which may be noted, namely, the importance of using the term "congenital" in the strict and accurate sense. The word is too often used to mean merely "present at birth," a use, or rather misuse, which leads to endless confusion of thought. If the deformity in these ribs was caused by an intra-uterine injury during any part of the gestation period, the deformity is an "acquired" one, and not congenital in any real sense. In other words it is a "somatic" character. If, on the other hand, the origin is to be found in the germ-plasm, it is a true "congenital" or "inborn" character; it is "germinal." It would conduce to much greater clearness if the terms "somatic" and "germinal" were used in this connection instead of "congenital" and "acquired," because so many use the word "congenital" in the loose sense of meaning anything present at the time of birth. The accident of the moment

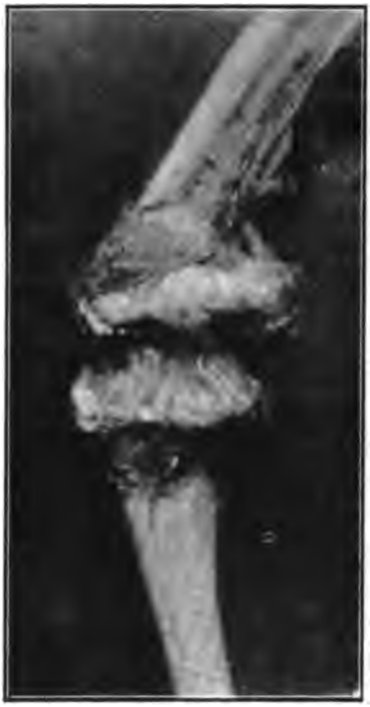

FIG. 2.

of birth has, of course, no connection with the origin of characters, and it should not be made the distinguishing basis. It would be just as accurate to speak of the result of an intra-uterine operation as "congenital."

I may add, that I have been unable to find any case on record of a similar nature to this one, and it is possibly unique.

SARCOMA OF THE SUB-LUMBAR LYMPHATICS IN A HORSE.

By W. H. BROOKE, M.R.C.V.S., Handsworth.

THE following is a short account of a case which is in my experience unique.

The subject was a brown carriage gelding, between fourteen and fifteen years of age, and standing about 16 hands high. Three-and-a-half years previously I had performed tracheotomy on it as a relief from roaring, which at that time quite prevented ordinary exertion; however, the insertion of the tracheotomy tube enabled it to work up to the time of the attack now to be described.

For six months previous to death (27th September) the animal had been in bad condition, and feeding precariously; there were no very well-marked symptoms, but, such as they were, they pointed chiefly to derangement of the intestinal functions. Towards the end of July the bowels began to be relaxed, and this symptom gradually increased in severity; indeed, during the last week of the subject's life the intestinal excreta were almost entirely fluid, and of an extremely unpleasant odour.

I examined the food, and enquired as to the regularity of feeding and watering, but I could detect no fault in this direction.

The molar teeth, which were irregular and sharp at the edges, received attention, and anodynes, astringents, hepatic stimulants, and tonics were all tried in turn, but gave only temporary relief. The purging meanwhile increased in severity; there were no symptoms of abdominal pain; the visible mucous membrane became more and more pallid.

The temperature during the last two months of life ranged from 100° to 102·3°; and, as the case seemed to resemble one which I had had under notice in a colt some two years previously, and which on *post-mortem* examination proved to be tuberculous, I decided to apply the tuberculin test. Accordingly, at 10.30 P.M. on 22nd September, the temperature being 102·3°, I injected 3 cc. of tuberculin (supplied by Prof. M'Fadyean). At 7.30 on the morning of the 23rd the temperature was 100°, at 10.30 A.M., 102°, at 1.30 P.M., 102°, and at 4.30 P.M., 102·3°. This result indicated that tuberculosis was not the cause of the trouble. As I had carefully watched excreta for parasites, and examined them microscopically for parasitic eggs or fragments, without result, I concluded that the cause must be some severe obstruction to the intestinal circulation; and, as there was an entire absence of hepatic symptoms, the circumstances seemed to point to lymphatic obstruction or tumour.

By this time (27th September) the animal had lost so much flesh and become so reduced generally that I told the owner that further treatment was useless; accordingly, the horse was destroyed the same day.

I made a *post-mortem* examination, and upon opening the abdomen along the middle line, the small intestines, which escaped first, were seen to be much shrunken; they contained very little ingesta, and were scarcely a third of the usual calibre. The large bowel was hardly half the normal diameter, and the vessels traversing it were extremely dilated and tortuous, containing blood of a deep venous tint. The vessel walls were so distended and thin that the slightest touch with the point of a knife penetrated them. Along the margin of the large bowel, following the course of the dilated vessels, were found quite a chain of tumour-like enlargements. Above the ileum, about 2 feet from its junction with the cæcum, I discovered a lobulated growth of encephaloid consistency, in weight about 4 pounds. Further, under the kidneys I found a great mass of tumour involving the posterior part of the root of the mesentery; this, on being removed, proved to be a little over 40 pounds in weight. It was irregularly spherical in shape, and on being cut showed a varied structure. In the centre of it were some lymphatic glands, the lobes of which were widely separated by the invasion of the tumour mass; and through it ran bands of a fibrous nature, interspersed with soft masses of an encephaloid consistency. All the enlargements were quite solid, without a suggestion of pus.

On returning from the *post-mortem* examination, I teased out portions of the growth finely, and placed them on a microscopic slide. This showed its structure to consist almost entirely of round cells with a small amount of fibrous tissue; some portions apparently fibrous on closer examination proved to consist of cells arranged in columns. By the kind assistance of my friends, Messrs J. Malcolm and Harris, sections were prepared and stained according to Van Gieson's method, and it then became manifest that the growth was almost wholly sarcomatous in structure and composed of round cells.

The extreme venous congestion was caused by pressure of the large growth upon the vessels uniting to form the portal vein. The liver was small in size, but apparently otherwise healthy. The thoracic organs were quite normal.

Reports.

RESOLUTIONS PASSED BY THE EIGHTH INTERNATIONAL VETERINARY CONGRESS, BUDAPEST, 1905.

INSURANCE OF CATTLE.

Insurance against Epizootic Diseases.

1. In the form and to the extent indicated in the reports submitted to the Congress, general compulsory insurance by owners against loss of cattle through epizootic diseases constitutes a simple, cheap, and workable State undertaking, provided it is based on co-operative action; and it is to be recommended wherever the State authorities do not accept and practise such compensation as a fixed principle, and where the agricultural population demand insurance.

Insurance against Death.

2. The insurance of cattle, swine, goats, and horses required for agricultural purposes against loss by death, accident, or compulsory slaughter, in order to safeguard that considerable part of the national wealth represented by the domesticated animals, should be regarded as a pressing duty of those representing agricultural and veterinary interests, and of the Government, as a means of furthering the rational breeding of domestic animals and promoting the prosperity of agriculture in general.

3. The simplest, cheapest, and most efficacious method of carrying out such insurance of animals is the formation of small local Insurance Committees, based on co-operative action, and the linking-up of such committees with a larger body, which would undertake the re-insurance of the local committees. Such larger undertakings should be State-aided, or, better still, be organised and conducted as Government or provincial institutions.

Local Insurance Committees, unattached to any larger body, should possess a uniform organisation, and be subject to control by the State.

4. The formation of insurance unions would awaken the agricultural population to the importance of general obligatory insurance, first of all, for cattle, which form the most important and valuable part of the holdings of domestic animals. Once the majority of cattle-owners were won over to this view, it would become the duty of the State or provincial authorities to institute and conduct general obligatory insurance.

Insurance of cattle could be followed by insurance of horses, swine, and goats.

5. The services rendered by private insurance companies, based on co-operative action, are worthy of recognition. Nevertheless, agreement as to the fundamental principles of insurance, the legal limitations in regard to the founding and management of such companies, and the subordination of insurance companies to Government control, is desirable in the interests of agriculture.

6. Insurance against loss in value of living horses as a result of prolonged and incurable disease or diseases limiting their power of work, as well as against accident, requires further development and extension.

7. Insurance of domestic animals against loss by death, accident, or compulsory slaughter, during transport on railways and ships, during exposure for sale at markets and other places, and when at grass on distant pastures, constitute a largely-felt want amongst breeders and cattle dealers.

Insurance of Animals destined for Slaughter.

8. Insurance of cattle destined for slaughter is a necessary corollary of the laws concerning inspection of meat. It would remove the difficulties entailed by the laws regarding meat inspection, and would prevent fraud.

While ensuring dealers in such cattle and consumers against losses due to meat inspection being thrown on their shoulders, it would protect individual members of the trade from excessive losses, and distribute such losses equally and in a modified form over the whole of the trade.

As insurance of animals destined for slaughter enhances the efficacy of the system of meat inspection, it also protects public health. This form of insurance is therefore in the interests of the meat trade, of agriculture, and of public health.

An interest which is so general justifies the foundation of general obligatory insurance of animals destined for slaughter by State and local authorities, and their support by State aid. The functions of such institutions should extend over as large an area as possible. In addition, the meat trade, which continues to expand, requires that individual institutions should come to a common understanding, and should co-operate.

9. The preparation and regular periodical publication of uniform statistics should be attempted by each variety of insurance.

Uniform statistics collated under similar systems would provide material for the scientific development of every form of cattle insurance, would enrich our knowledge of the diseases of domestic animals, and would lay the foundation for the development of veterinary-pathological geography.

10. Until this question of insurance of cattle is completely cleared up, it should receive a place in the order of the day for every succeeding International Congress.

Uniform Scheme for Periodical Veterinary Sanitary Returns.

The VIIIth International Veterinary Congress in Buda Pesth recommends the preparation of a uniform scheme for periodical veterinary reports, both in the interest of the veterinary officials of individual states and for the purpose of controlling international trade in cattle; it commissions its permanent staff to take the necessary measures for this purpose.

The Relationship between Tuberculosis in Men, Cattle, Birds, and other Domestic Animals (especially Dogs).

The Congress is of opinion :—

1. That bovine tubercle bacilli are infectious for man, that some human tubercle bacilli are very dangerous for cattle, and that in certain cases reciprocal infection is possible.

2. That it is desirable further to investigate to what degree avian tuberculosis constitutes a danger for man and mammals.

3. That under any circumstances it is indispensable to continue precautions against the danger which may result from infection of man by bovine tuberculosis.

The Struggle Against Tuberculosis in Domestic Animals.

1. The struggle against tuberculosis in cattle is urgently necessary, not only on account of the economic losses associated with the disease, but also on account of the danger of human infection.

2. The stamping-out of tuberculosis in cattle by the owner (voluntary action) is feasible, and should be generally attempted. It calls for the earliest slaughter of dangerously-infected animals, and sedulous precautions against the infection of calves and other healthy animals. The voluntary stamping-out of tuberculosis in cattle can be assisted by the State through the propagation of

enlightened views as to the nature of tuberculosis, the method of infection, and the significance of the tuberculin test, as well as by the assistance from State funds.

In connection with the struggle against tuberculosis in domestic animals, tuberculin appears to be the best diagnostic means yet discovered.

The sale of tuberculin should be controlled by the State. Under no circumstances should tuberculin be supplied to others than veterinary surgeons.

3. It is extremely desirable that the struggle against tuberculosis in cattle should be undertaken by the State. If entered on with certain precautions it is quite feasible, and it would check further spread of the disease and gradually bring about its complete cessation.

Such action would necessitate:—

(*a*) Compulsory notification by veterinary surgeons of every case of tuberculosis met with by them in practice.

(*b*) Early slaughter of dangerously-diseased animals, particularly those with mammary, uterine, or intestinal tuberculosis, or with extensive tuberculosis of the lungs; compensation from State funds, and interdiction of the return by co-operative dairies of skim milk unless after sterilisation.

Protective Inoculation Against Tuberculosis in Cattle.

The VIIIth International Congress urgently begs the various Governments to provide the means for carrying out on a large scale experiments directed towards testing the value of protective inoculation against bovine tuberculosis under the varying conditions existing in agricultural practice.

Until, however, the exact value of protective inoculation is finally determined, the continuance of the sanitary precautions already proved to be of such value is necessarily called for.

Uniform Principles for Estimating the Tuberculin Reaction.

1. The preparation and supply of tuberculin should be controlled by the State.

2. No animal whose temperature exceeds 39·5° C. is a fit subject for the tuberculin test.

3. A rise of temperature to above 40° C. in any animal whose temperature at the moment of injection was below 39·5° C. is to be regarded as a positive reaction.

4. Any rise in temperature between 39·5° and 40° C. must be regarded as of doubtful significance; animals exhibiting such require special study.

Uniform Principles Regarding the Mallein Reaction.

1. Unless the results following the injection of mallein exhibit the characteristics of a typical reaction, they must not be regarded as indicating the existence of glanders.

2. A typical reaction comprises a rise in temperature of at least 2° C. The rise would extend above 40° C.; during the course of the first day the temperature curve usually exhibits a plateau or two peaks, and on the second, and sometimes even on the third, a more or less marked rise. This rise in temperature is accompanied by a local and general reaction.

3. Any rise in temperature which falls short of 40° C., and higher atypical reactions, necessitate a second test.

4. A gradual rise of temperature, sustained for some time, is indicative of glanders, even though it differs from the ordinary type of diagnostic reaction.

5. The local typical infiltration at the point of injection is a certain indication of glanders even when the rise in temperature and the general organic reaction fail.

6. Animals which have undergone the mallein test, whether or not without

reaction, should always be tested a second time after an interval of ten to twenty days.

7. The preparation of mallein should only be entrusted to scientific Government institutions or to institutions recognised and controlled by the State.

8. With the object of determining the full value of mallein, and of clearing up many still-unexplained points in regard to the mallein reaction, the Congress requests the various European Governments to appoint committees to study the question.

Protective Inoculation Against Foot-and-Mouth Disease.

1. Experience shows that veterinary-police measures give excellent results if undertaken immediately on the outbreak of foot-and-mouth disease, and if conducted by veterinary surgeons.

2. It is desirable that the different States should undertake to notify each other by telegraph of the first appearance of the disease in their various possessions.

3. Having regard to the very interesting results of protective inoculation, the Congress expresses a wish that the various Governments would assist in the study of its practical applications.

Measures against Swine-Fever and Swine-Plague. Protective Inoculation.

1. Swine-fever (which is often a mixed infection) ought to be controlled by veterinary-police and hygienic measures.

2. At the present time no known protective or curative inoculation methods can be recommended. Further careful investigation is necessary to determine whether such methods and materials are to be sought in the directions already indicated, or whether entirely new methods must be devised.

Measures against Rabies, including the Stamping-Out Method.

1. The struggle against and stamping-out of rabies in a Continental State can only prove successful when appropriate veterinary-police measures are strictly carried out in all the neighbouring countries. It is therefore urgently necessary that, as regards rabies, the work of the veterinary police should be undertaken in all countries simultaneously and on the same principles.

2. The duty of reporting, which up to the present has only been compulsory as regards rabid dogs and dogs suspected of rabies, must be extended to include all animals which have been bitten by rabid or suspected-rabid dogs. Not only must the owner of the animal and the persons mentioned in par. 9. of the German Contagious Diseases Act be required to give notice, but all those who have knowledge of animals which have been bitten by such dogs.

3. It is an open question whether it might not be desirable to extend the muzzling order over a larger area than hitherto, and for a longer time than three months.

4. It is desirable that a uniform law relating to the ownership of dogs, and containing the following conditions, should be introduced into all countries and severely enforced.

(*a.*) All dogs, both in towns and in the country, shall be reported, registered, and taxed.

(*b.*) Every such animal shall be provided with a metallic ticket attached to the collar and bearing the name of the owner and the number of the dog in the tax list.

(*c.*) Dogs without such metallic tickets and without muzzles shall be taken charge of by the police, and if not claimed within a stated time shall be killed.

Tropical Diseases.

Tropical diseases should form a subject for special instruction in the veterinary schools of tropical countries and of countries possessing tropical colonies.

The individual Governments should investigate the sanitary condition of the domestic animals in their colonies, and especially enquire into the existence of diseases produced by protozoa.

It is desirable that the geographical distribution of different infectious diseases, and the general laws relating to their spread, should be defined.

In every colony a Central Institution for Parasitology should be founded, provided with ample means and a technical staff of specially qualified investigators in bacteriology, mycology, parasitology, and entomology.

In colonies which are not yet provided with a veterinary-police staff, such a body should be constituted. This staff would control the operations of experts appointed to deal with disease; it should possess the most extensive possible powers for combating infectious diseases.

The institute for parasitology, the sanitary police staff, and the staff for dealing with epizoötics should possess the most complete autonomy, and should only be answerable to the highest civil power.

Finally, the scientific and administrative officers of these different institutions should pass their entire careers in the same colonies, or at least in colonies comprised within the same geographical region, inasmuch as success can only follow methodical work by those accustomed to the sanitary conditions of a given country.

Milk and its Treatment, with Especial Reference to the Observance of Hygienic Requirements in Milking.

1. Milk hygiene, inclusive of a practical course in milk bacteriology and police control of the milk trade, should form a portion of the curriculum of veterinary colleges.

2. The use of such terms as "tubercle-free milk," "milk for children," "hygienic milk," etc., as advertisements, should be forbidden, and the preparation of milk so described should be subject to special control (constant supervision of the animal's health, feeding, and treatment, of the methods of obtaining the milk, and of its cooling immediately after withdrawal).

3. The police regulations should contain a clause prohibiting milk containing dirt from being offered for sale. In practice the milk to be examined would be placed in a litre bottle of clear glass. No deposit should occur after standing for three hours.

Adulteration of Meat and Meat Products and the more Recent Methods of Detecting such Adulteration.

The Eighth International Veterinary Congress is of opinion that only such materials as are known to be harmless, like salt, nitre and sugar, and such methods as smoking, should be used for the preservation of meat and meat preparations. The use of any other substances to preserve or colour the said provisions should be forbidden: firstly, because they are added for purposes of deception and to give a more attractive appearance than the substance would naturally present, and, secondly, because it is uncertain whether or not they may prove injurious to health.

Feeding with Molasses.

The biological section of the Eighth International Veterinary Congress is of opinion that molasses is an important form of food, both from the economic and dietetic standpoints. The choice of a material for absorbing the molasses must be based on physiological and economic grounds.

Organisation of Future International Congresses.

1. International Veterinary Congresses tend to advance all branches of veterinary science and to assist their practical applications.

2. The programmes of the Congresses should include special sections, amongst others a clinical, a tropical, and a physiological anatomical section, in addition to a number of general meetings of all the sections.

3. A standing International Congress Committee should be chosen to which the following members of the present Committee should be elected.

(*a*) The two chief officers of the present Congress.

(*b*) Two representatives of the country in which the next Congress shall be held.

(*c*) A delegate from each of the other countries which have hitherto been well represented.

4. The first duty of this Committee should be to formulate a plan of organisation and a thorough scheme of work to be carried out at the next Congress. This scheme to be submitted to the last meeting of the next Congress for final approval.

5. Until further action is taken, the Committee will arrange its own order of business and elect a president and general secretary. Voting will be by ballot, a simple majority to decide.

6. The Committee will also fix the "orders of the day" for the next Congress, and, before the date of the latter, ascertain whether the Jubilee Congress of 1913 can take place in London.

7. The IXth Congress will meet in Holland in 1909.

Promotion to the Degree of Dr Med. Vet.

1. The Congress considers it necessary that the veterinary teaching establishments, whether faculties or independent colleges, should have the right of conferring a degree of Doctor of Veterinary Medicine.

The Congress considers it desirable that the degree of Doctor of Veterinary Medicine conferred by the faculty of a university, should carry the same "kudos" as degrees conferred by any of the other faculties.

3. The Congress desires the standing Committee to take the necessary steps for the attainment of this object. (*Berl. Tierärztl. Wochenschr*, 1905, No. 41.

ROYAL VETERINARY COLLEGE, LONDON.

INAUGURATION OF THE WINTER SESSION, 1905-1906.

THE winter session at this institution was opened on Monday, 2nd October, when a large audience, comprising the teaching staff, past and present students, and members of the Board of Governors, assembled in the large lecture theatre of the College to hear the Inaugural Address. The Chair was occupied by Sir THOMAS ELLIOTT, K.C.B., Secretary to the Board of Agriculture and Fisheries.

The CHAIRMAN said that his first, perhaps his sole duty, that day was to call upon Mr HUNTING to deliver the introductory address. There was no more hearty lover of veterinary science than Mr Hunting, and he was quite sure that the words which he would utter would receive their careful attention, and that all would profit by what he had to tell.

Mr W. HUNTING, F.R.C.V.S., who was received with applause, then delivered the introductory address, as follows:—

"MR CHAIRMAN AND GENTLEMEN,—My first duty is to express the feelings of pride and pleasure with which I undertake the task permitted me to-day. I am not a teacher, not even a graduate of this school. I am an ordinary London practitioner, and I can only interpret my presence here as a mark of the goodwill and confidence existing between the Royal Veterinary College and the Metropolitan practitioners.

"My next duty is to refer with sincere regret to the loss sustained by this Institution through the death of General Sir Frederick FitzWygram. He was, and had been for years, a Governor of the College, but he was no ordinary governor. He held a veterinary diploma and had been President of the Royal College of Veterinary Surgeons. He was an enthusiast in veterinary work, and although engaged in all the multitudinous affairs inseparable from his high military, political, and social position, always found time to promote the advancement of veterinary science. To this School and to the Royal College of Veterinary Surgeons he freely gave his time and thought. One of the last acts of his life was to found a handsome prize, open to all veterinary students—a prize which I hope more than one of you may be able to secure. It will be long, I fear, before such another friend of the veterinarian is found either here or at Red Lion Square.

"And now, gentlemen, I propose to ask, and attempt to answer, two questions which are of great importance to you:—What have you come here for? What are your prospects when you leave with a diploma?

"In reply to the first question you would all probably exclaim—to obtain the diploma! But that is not enough. Have you come *determined* to get it? and to be in possession of it by the end of your fourth session? If so, and if you have the natural aptitudes necessary to the making of a good veterinary surgeon—love of animals and some knowledge of their habits, love of science as applied to life, and a healthy curiosity concerning the causes, prevention, and treatment of disease—then you have chosen wisely in coming here.

"If, on the contrary, you are obeying an impulse suggested by the comfortable appearance of some practitioner whom you have known only in fine weather, strengthened perhaps by the possession of sporting or horsey tastes, you may encounter the keenest disappointment, and live to wish you had followed some other calling. In a still worse case is the man who is here without any very strong inclinations, who would like to have a diploma if he could get it without much effort, who would just as soon follow some other calling if it promised as good an income and as pleasant a life. Unfortunate, too, is the man who looks upon the science he has to learn and the art he has to acquire as obstacles to his commencing business instead of as essentials to his profession and additions to his power of using and enjoying life. But I will suppose that all here look forward with pleasure to the acquisition of knowledge and only desire a diploma as a mark of its possession, and I again ask why have you come here? there are other schools. Probably most of you acted upon the advice or order of parents and guardians. I am pleased to be in a position to say you were well advised. No other school can offer you quite all the facilities to be found here. No other school possesses a better staff of teachers or a more complete material equipment. Not one can offer such an extensive and varied clinique for your practical training. It only remains for you to honestly use what is so lavishly provided.

"My second question is one on which I neither desire to dogmatise nor to prophesy. I propose to briefly review the past of the veterinary profession, notice its present state and then with the information afforded by this enquiry attempt a reasonable prognosis as to its immediate future.

"*The Past.*—It is over a century since this school was founded. Veterinary science had then made some little progress in France, and the first professor at this institution was a Frenchman who had been educated at a French veterinary school, Unfortunately Sainbel did little more than establish the college. He gave his first lecture in 1792, and fell a victim to glanders in 1793.

"At that period some form of veterinary art existed and was practised by farriers and empirics. Only a few practitioners could boast of the least scientific training; but the works of Gibson, Clark, Osmer, and others are evidence that practical observations had been made and recorded. A few practitioners there were who had previously studied human medicine, and one at least who had taken a course at the Paris Veterinary School, so we may believe that the learning of the doctors and surgeons had at least tinctured the methods of veterinary practice Still the practice of medicine and surgery amongst the domestic animals at the end of the eighteenth century was in a deplorably low state. Horses were doctored by the groom or the farrier. Cattle and sheep received litt'e attention save that afforded by the owner or attendant. Dogs were entrusted to the gamekeeper. Army horses obtained their only treatment from the farrier or riding master save when the regimental doctor was a sporting man and gave his more trained assistance.

"The arrival of Sainbel in England was timely, as the neglect of sick animals and the losses due to disease had inspired many owners of stock with the urgency of securing better advisers. The Odiham Agricultural Society had framed a scheme for sending two young men every year to the Paris school for study, and only dropped the plan when Sainbel suggested the establishment of a school in London.

"A strong committee of noblemen and gentlemen, among whom were some of the leading medical men, was formed, and in 1791 the veterinary college was established. That the committee was in earnest is shown by the success which attended their efforts after the death of Sainbel, for it was then almost impossible to replace him. For a time the duties of the school were carried on by selected students; and John Hunter, the immortal anatomist and physiologist, who had from the commencement assisted and was a vice-president of the school, came to the rescue and arranged for students to attend gratuitously the lectures of several of the leading teachers at the London Hospitals. This free entry to the classes of medical teachers was continued for many years and partially supplied the want of tuition in the college on important subjects. We must ever be grateful to the men who thus helped the start of our struggling profession, but we must also acknowledge that it was only a make shift arrangement requiring improvement. Every subject taught men preparing for a special calling is best taught by experts in that calling and by a curriculum specially arranged for their special wants, conditions now existing in this college.

"A year after Sainbel's death, in 1794, Coleman, a rising young surgeon, pupil of Hunter and friend of Cline and Astley Cooper, was appointed professor. By his energy and genius he soon achieved success and established the college on a sound but narrow basis. I say narrow, because the teaching was limited to equine physiology and pathology, and the horse was the only patient in the hospital, as Coleman was the only teacher for the first four years. The medical profession continued their support, and formed an examining body which gave a certificate to all proficient students. Coleman possessed not only great natural talents, but influential friends, and early in his career was appointed veterinary surgeon general to His Majesty's army. As the result of his advice and direction, it was soon acknowledged that the health of the horses had improved, and the losses from pneumonia and glanders markedly declined. From a list of the *The Examined and Approved Students of the Veterinary College* published in 1802, we gather that between 1794 and 1802, 102 men had obtained certificates, and of these thirty-four had obtained appointments in the cavalry. This was a great triumph for Coleman, and a full recognition of the value of his pupils. But the professor did not stop at obtaining appointments; he gained for his men the King's Commission, and so contributed to the social position of the newly trained practitioner in the army, and indirectly to those in private practice.

"A further development of veterinary teaching took place in 1823, when William Dick, a graduate from the London college, opened a school in Edinburgh. This soon became a success. He had the support of the medical men in the town, and the Highland and Agricultural Society assisted to form an examining board and granted certificates to proficient students. Thus veterinary science obtained another centre from which to expand, and recognition was given by public bodies in Scotland and in England.

"In 1831 Coleman was elected a fellow of the Royal Society, and his name remains, I believe, the only one representing veterinary science upon the roll of that learned and exclusive body. The honour was well deserved and not too soon in coming. Coleman was now sixty-six years of age, and losing some of his energy but none of his masterfulness. He continued his teaching but opposed all assistance or alteration in his works.

"The number of practitioners who had passed through the school was large, and constantly increasing. The school was the fountain from which they all sprang, and in which they were all interested. They not unnaturally looked for some progress, and suggested the addition to the examining board of a member or two possessing practical knowledge of veterinary science and art. They also wished to have a veterinary surgeon on the governing board of the college, but all their demands were refused. The spirit of discontent increased, two periodicals were started, an association was formed, and some of the practitioners added useful volumes to our scanty literature.

"On the death of Coleman in 1839 the opportunity occurred for widening the course of study at the college and increasing the teaching staff. To some extent this was done, but it was not till 1842 that the curriculum included animals other than the horse. The year of Coleman's death was the year in which foot-and-mouth disease invaded the country, and the rapidity of its spread drew immediate attention to its dangers. The Agricu tural Society of England put itself into communication with the college, and after a year or two a Chair of Cattle Pathology was established, and Prof. Simonds added to the staff. This was an important event both for agriculture and for veterinary science. Without the assistance of the society it would have been difficult for the school to have used the opportunity for the study of cattle diseases, and without the school the society could have done little. The two working together occupied a grand field for observation and research, and I think I may add, one that has well repaid both agriculturists and veterinarians. About this time another disease was introduced from the continent, pleuropneumonia, and a few years later stock-owners had to contend with a third animal plague, sheep-pox, all of which occupied the attention of Prof. Simonds for many years.

"Shortly after the establishment of the Chair of Cattle Pathology at this school an important event was brought about by the general body of practitioners. For two years they attempted to obtain a Charter of Incorporation, but met with opposition. In 1844, after arranging with the veterinary colleges in London and Edinburgh, they succeeded in obtaining the instrument which gave them a legal organisation under the name of the Royal College of Veterinary Surgeons, which recognised them as a profession and provided that all future candidates for admission to the profession should be examined by a Board appointed by the Council of the Royal College of Veterinary Surgeons.

"At the time of our incorporation veterinary science laboured under the disadvantage of having few good text-books. Neither in the schools nor in the ranks of the practitioners were many men with literary abilities, and a still smaller number were those who read any language but English. The result was that the work done by the numerous Continental schools was not generally available, and this was a great loss to us. Until Percival, who read French, published his Anatomy, our students had little help from books, and the same author gave us the first systematic text-book on equine medicine and surgery when he wrote his Hippopathology.

"In 1865 occurred an event which made perhaps a greater impression on the veterinary profession than anything since the founding of the Royal Veterinary College, viz., the invasion of England by the cattle plague. This dreadful disease was introduced from Europe at two or three different ports and had a firm hold on our cattle before it was recognised. In fact only three men had seen it. Simonds and Ernes, who had been sent in 1857 to investigate it on the Continent, and John Gamgee who had seen and studied it abroad. To the latter must be given the credit of first recognising the serious nature of the disease and of most loudly appealing to the authorities and the public to take immediate and active measures for its control. Gamgee, who was Principal of a new veterinary school in Edinburgh, had for years warned his students and the profession of the probability of the cattle plague being introduced, of the loss it would cause, and of the stern measures necessary for its extermination.

"To Prof. Simonds, however, fell the task of officially advising the Government, and to him and Prof. G. T. Brown was allotted the work of directing the measures which, after much loss of time, were enforced by Government. These measures were compulsory slaughter of all diseased animals and those in contact with them, restriction of movement in infected areas, and compulsory notification by the owners.

"The disease was first recognised in London on 4th July 1865. It was reported by Simonds to the Privy Council on 10th July, but it was not till 18th August that an order was issued giving some powers to Local Authorities. In September a Royal Commission was appointed to enquire, but not till 20th February 1866 had Parliament passed the Cattle Diseases Prevention Act, under which the disease was stamped out. At the time of the passing of this Act 18,000 cases were reported to have occurred in the previous week. Success attended the efforts of the Privy Council, and cattle plague was stamped out by the end of the year. It cost the country £6,000,000. During the first six months, when practically nothing was done, the press teemed with articles and letters abusing the veterinary profession for having nothing better to suggest than slaughter. The medical profession took an active share in offering suggestions—pathological and therapeutical—and in opposition to the veterinarian. The result established our reputation as knowing our own business better than anyone else, and gave us a position as professional men and scientific experts which has since only been added to and never denied—except perhaps by a late Minister of Agriculture.

"The awful losses caused by the cattle plague accentuated the dangers from other diseases of animals, and the success attained in stamping out the plague encouraged the Government to attempt further control of contagious diseases. In 1869 Parliament passed the Contagious Diseases (Animals) Act, under which were scheduled cattle plague, pleuro-pneumonia, foot-and-mouth disease, and glanders. It was a step in the right direction, as shown by the rapid suppression of two outbreaks of cattle plague which occurred in 1872 and 1877. The Act has since been frequently amended and extended, notably in 1878, and again in 1894, when the title was changed to the Diseases of Animals Act.

"From 1871 to 1901 the work of the Veterinary Department of the State was directed by Professor, now Sir George Brown. His work looms as large in veterinary history during the latter half of the nineteenth century as did that of Coleman during the early part. We all know him as a distinguished man of science; and a Minister of State has left on record that "Brown was one of the best organisers, if not the best, he had met in his long experience of government departments." As an instance of his judgment and sagacity it may be mentioned that almost his last official dictum was—" If you wish to stamp out swine fever you must enforce cattle plague regulations."

" Slowly and gradually under the efforts of legislation, assisted by the private practitioner, animal disease has been controlled. Foot-and-mouth disease, which had prevailed since 1839, was exterminated in 1886, and pleuro-pneumonia in 1898. Unfortunately much of our legislation was timorous and nearly all permissive. A great deal of it was left to be carried out by Local Authorities, and was neglected. Each disease that has been taken over by the Central Authority for active suppression has been successfully dealt with except swine fever. But it must be noticed that the Central Authority has always had greater powers and more rigorous restrictions than were ever given to the Local Authorities. Of course I know that in a country of representative Government and party politics it is not easy to make laws which embrace all that experts would consider necessary for speedy and certain success. Every new regulation causes friction and disturbs some vested interest or popular prejudice. The result is our laws are compromises and require frequent amendments to bring them up to the standard at which they are most useful.

"The Department that enforces the law is only an administrative body, and naturally evades as much as possible the opprobrium which accompanies any full use of its powers. Fear of failure seems to be a dominant note in departmental action. Success is the only thing which justifies strong and logical effort, and until that success is attained there is always a party of opposition which makes itself heard and frequently interferes with the best-directed measures. There was howling opposition to the regulations which stamped out cattle plague. There was such a determined stand against the restrictions which cleared the country of foot-and-mouth disease as for a time to threaten a cessation of the work—but a little firmness ensured success. Quite recently the Board of Agriculture has been successful in ridding the country of that dangerous plague, rabies, but not without having to face a most active campaign of ignorance and lying. This year an earnest effort has been commenced to control sheep-scab, and the very first action of the Board resulted in an organised opposition by the men who are largely the cause of the dissemination of the disease.

" Although one may sympathise with the Central Authorities in their struggle against unintelligent and selfish opposition, I cannot but think that a little more thoroughness and a little less timidity might mark their action. " Grasp the nettle firmly," and " Don't make two bites at a cherry," are bits of proverbial wisdom which force themselves upon us in this connection. Precedent is all in favour of well-directed firm measures; they have in the past led to success though always accompanied by opposition and difficulties.

" There is one scheduled disease which the authorities have much neglected —glanders. And the curious thing about it is that there is no active opposition to more thorough measures—they have been invited and prayed for. Glanders differs from other scheduled diseases in that it only attacks horses and men. There are no powerful societies directly interested to agitate and bring pressure on Parliament or the Department. It is not public opposition which prevents adequate regulations being framed; on the contrary it is apathy and want of knowledge. The general public knows nothing of diseases of the horse, and the horse-owner is, *per se*, an isolated person with no organisation to voice his grievances. When we plead that glanders not only destroys thousands of horses every year but a number of human beings we are referred to the Registrar General's returns, and told that the mortality is only two men per annum. But the mortality returns are not true. Probably fifty or a hundred human beings are killed annually by glanders, but their death is registered as due to other diseases. During the last two years a large increase in the number of diagnosed cases is apparent, and the explanation is that the bacteriologist has been called in and makes the diagnosis—usually on the day of death or before the inquest. Never is a case registered by a private practitioner; it is only from public hospitals they are heard of, and yet it is obvious that private practitioners must see more cases than the hospitals. The fact is that the larger number of cases of human glanders are registered as typhoid-fever, influenza, rheumatic fever, or blood poisoning. I am not reflecting upon the skill of the medical profession. I believe glanders in man to be usually undiagnosable without the bacteriologist, and I should welcome the co-operation of the medical profession in an effort to obtain measures for stamping out this deadly plague. Their help was invaluable in the case of rabies, and so it would be with glanders. I firmly believe that the eradication of glanders in horses would save more human life than did the extinction of rabies in dogs.

"The services rendered to the State by veterinarians have been great, and they have been recognised. Some plagues have disappeared, others are being dealt with, but there are more to tackle. Tuberculosis, contagious abortion, mange, strangles, and equine pneumonia are all diseases which can only be controlled by a wide collective action, and I believe that such action would give a good economical return for the labour and cost. In time, and as other plagues are successfully dealt with, those I have mentioned and more will probably be scheduled.

"The services rendered to the Army by veterinarians were long ago acknowledged and since have been more fully recognised. It seems to me, speaking as a civilian, that hygiene, sanitation, and preventive medicine offer ever widening fields of usefulness to veterinary officers—provided the army powers would give increased authority and responsibility to the men most capable of directing such matters. Quite recently the Army Veterinary Department has succeeded in having removed some little grievances, and now the alterations of rank, title, pay, and pension have given satisfaction to its members and increased the attractions of the department. I cannot but think the army offers a fine career to young veterinarians and that it should be more largely taken advantage of.

"Municipalities and County Councils have during the last twenty years increased their appreciation of veterinary science and utilised the services of veterinarians more fully and on wider lines. Local Authorities who keep horses not only employ a veterinary surgeon to treat the sick and lame animals, but in many cases entrust him with the feeding and general management of the stud. The advantages and economies resulting to the ratepayers are made evident in such places as Liverpool, Manchester, Birmingham, and Sheffield. Slowly, too, it is being recognised that the trained expert who understands animals in health and disease is the fittest person to inspect the places in which they are kept and the products which they yield—meat and milk. The fact that there are diseases transmissible from man to animals, and from animals to man, renders it necessary to establish co-operation between medical and veterinary officers for the protection of the health of man. To leave the matter merely in the hands of either is inadmissible. Great hardship has been inflicted on stock-owners by proceedings taken in error by a medical officer, and great dangers to man have resulted from inefficient veterinary inspection.

"One more field of increasing veterinary work I must briefly refer to, viz., that in our Colonies and dependencies. In Australia, New Zealand, and South Africa a number of veterinarians have for some years been engaged by the respective governments in investigating and preventing disease among animals. Nothing in our annals is more satisfactory than the success which has attended their efforts, and the hearty recognition which has been made of their services. In India the Government has established a Veterinary Civil Service which is doing good work, and promises an increasing opportunity for those engaged in it. Other dependencies, though on a smaller scale, have seen the necessity for trained men to look after their animal wealth and have appointed veterinary surgeons.

"*The Present.*—From this superficial glance at our history I think it must be evident that the veterinary surgeon has constantly found an ever increasing sphere for his usefulness, and that he has always risen to the requirements of the situation. A century ago his studies were short and not very deep. Of his own initiation his period of study has been gradually increased and the range of subjects extended. His preliminary education which at first was ignored has now been fixed at the same standard as that required by other liberal professions.

"We have discovered that curative medicine is not the sole end and aim of our calling, but that hygiene and preventive medicine are of equal if not more importance. Owners of stock have noticed the great improvements and economies which have followed the appointment of veterinary surgeons to positions where a knowledge of physiology and pathology gave them untold advantages over the man who possessed no such knowledge.

"Our College and schools have provided that new fields of work shall not lack trained applicants, and hygiene is an important part of the curriculum at every school. The four-years course of study, which is now compulsory, is sufficient to enable a man to obtain a license to practise, and his diploma is a guarantee that he may be trusted to practise his art and science with advantage to his client. In veterinary science, as in all others, the minimum of proficiency required must always fall short of what the ablest men demand. To meet this requirement post graduate courses of study have been established, and it is really remarkable how much this finishing touch of College education does for its votaries.

"The study of disease is ever advancing, and one can imagine no end to the beneficent discoveries which will certainly be made. Comparative pathology has already elucidated problems which a few years ago were not considered within practical solution. It offers such probabilities of controlling, preventing, and curing disease in men and animals as were once only dreamed of. The use of sera, vaccines, and toxins for the prevention and cure of disease is daily developing. Extracts of animal organs are in constant therapeutic use, and yet we are only on the threshold of this new domain. It is safe to say that we are on the eve of pathological discoveries which will eclipse in value anything which has been done before, and that these discoveries will be as applicable and useful to veterinarians as to medical men. Comparative pathology covers a vast sphere—the whole animal kingdom, and requires workers in all its divisions. Veterinarians have already contributed a fair share to the general fund of knowledge, but we are hampered by material and financial considerations. Our field of work is as large and important as any other, and our men are able and willing, but research brings no immediate return, and we are poor. What we require is some endowment, and our claim should be pressed upon the Board of Agriculture. If the Board endowed veterinary research its action would not be a one-sided gift—what it sowed it would reap tenfold. We want our own laboratories for veterinary pathological research. Ours is not a subsidiary division of pathology—it is a part of the whole science, and a special and well marked branch more easily and profitably followed by those who have had previous training in the peculiarities and diseases of animals. I venture to say that the founder of the Brown Animals Institute would agree with me that an institution dedicated to the study of pathology and the relief of animal sufferings, such as exists as Vauxhall, would be more reasonably and successfully conducted by a veterinary surgeon than by a professor of human medicine.

"We have in this School a pathological laboratory capable of fully supplying the wants of the students and providing for the post-graduate workers who have been invited to share its benefits. We have, too, in Prof. M'Fadyean a scientist whose research work is known and appreciated all over the world; but we want further opportunities ahd more workers to enable ns to reap the rich harvest standing in the field of veterinary pathology.

"*The Future.*—Now, gentlemen, I have completed my brief sketch of our professional development, and must try to frame an answer to the question—What are your prospects?

"We have seen the profession grow and expand, we have seen fresh duties imposed upon it, and always with the result that they were successfully grasped. Is it possible that we have reached our zenith and that our usefulness will now decline?

"There are pessimists who tell us that the ever increasing importation of dead-meat will cause diminution in our home live-stock; that the advent of electric and other motors will drive the horse off our streets and fields; that the onward progress of legislation and preventive medicine will so decrease the prevalence of disease that there will soon be little to treat; that the multiplicity of veterinary schools will so inflate the numbers of the profession that a time will shortly arrive when we shall be the most overcrowded of callings.

"Gentlemen, I believe none of these things. The influx of dead meat, by cheapening food, may probably affect to some extent the growth of our herds, but the demand for milk and butter which is now far ahead of supply must lead to increase of our dairy stock. The pedigree stock of this country is the best in the world, and for some generations to come will be required by all the great grazing countries to grade up their herds. Without our pedigree animals foreign countries cannot obtain the quality of beef and mutton necessary to win a place in our markets. Pedigree stock is more valuable than common sorts, and the greater the value of animals the more rapidly does the owner seek veterinary advice.

"As to the motors that are to displace the horse, I allow that electric tramways are a serious competitor. Probably 20,000 horses have already been displaced in Great Britain by electricity, and more are sure to follow. The motor car has also displaced a few horses, but in many cases the owners have returned to the animal. The only motor car which at present permanently competes with the horse is the one used for fast work and the carriage of a few people. The motor omnibus and the motor lorry have yet to develop considerably before they will displace horses. Their first six months work, whilst the machinery is new and everything sound and taut, has deceived people into believing that their primary condition is permanent. Owners will have a rude awakening and be glad to resume horse haulage. Probably in time engineering skill will surmount all difficulties and the inanimate motor be a practicable and economical article—but that time is not yet. Even when the motor takes its place as a reliable carrier there will be men and women who prefer to ride or drive the horse. There will be racing at Epsom and Newmarket, and hunting with the Quorn and the Pytchley. The stag, the fox, and the hare will at least have to go before the horse is discarded. South Africa has shown that the horse is still a military necessity, and we may rest assured that the Army will require veterinary surgeons for a long time to come.

"Those who fear the curtailment of veterinary practice by legislation successfully stamping out diseases of animals must be very sanguine admirers of the

Board of Agriculture. As the Board passes from success to success it must naturally keep adding to the schedule of diseases, and the list is long. Besides, every disease successfully mastered only enhances the value of veterinary work and attracts owners of stock to make greater use of it.

"The question of over-crowding as the result of an excessive number of graduates pouring out of the multiplicity of schools is imaginary. At present the annual increase of members is very small, and I am unaware of anything which should suggest that we are likely to witness a rush for admittance to the veterinary profession. I am told that there are vacancies in the Army and that good assistants are very scarce.

"There is still another bogey that is brought up to frighten intruders and to discourage young men. It usually is put in the form of a question—What are we going to do in the face of the ever-increasing sale of quack medicines and the mendacious advertisements by which they attract purchasers? Well, we can do nothing. except hope for the enlightenment of stock owners, and perhaps lend a helping hand to spread knowledge. We suffer from quacks owing to the errors of our forefathers. For centuries it was the custom for medical and veterinary practitioners to assume a mystery in connection with drugs and an omniscience in relation to disease that has misled the human patient and the owner of domestic patients. The public has acquired a notion that each disease has a specific cure, and that something in a bottle is necessary and will probably be efficient. What may be in the bottle or pill is to them quite immaterial so long as some printed assurance is given with it. This simple faith in advertised preparations will probably exist as long as men have little scientific education, and are taught blindly to trust in authority. We must not assist the exaggerated belief in drugs, but endeavour to impress the value of our diagnosis and personal attention. Make a fair charge for professional attendance and avoid over charges for medicines.

"I do not, then, believe that there will be much shrinkage in the sphere now occupied by our ordinary practitioners. I look forward very confidently to a large increase in other directions—to growth in hygienic work and expansion in classes of patients. The smaller domestic animals kept as pets and companions now form part of most households, and afford ever-increasing opportunities for veterinary practice. Their diseases are more varied than those seen in the horse and ox, and are of the greatest pathological interest. I look forward also to a great increase in the number of appointments which must necessarily accompany the growth of hygiene and preventive medicine. Every large town will soon have a veterinary officer, as they now have a medical officer. The inspection of abattoirs, dairies, and cowsheds must inevitably be entrusted to veterinary surgeons as the authorities become aware of our special fitness for such posts. There will be both in this country and our Colonies increased demands for men capable of conducting research work. The prevention of disease requires knowledge of its nature and method of spread. There are few diseases about which we know enough to render our preventive action certain, and therefore I anticipate that research in comparative pathology will be insisted upon by the State and by Local Authorities, who, of course, will provide the necessary accommodation, and appoint the most useful men to conduct the work.

"And now, gentlemen, I hope I have adduced sufficient data to enable us to answer the question—What are your prospects on leaving this school with a diploma? In my own mind no doubt exists. The veterinary profession has not yet reached its zenith. It will continue its development, and afford its members an interesting and useful career. It will rise yet higher in public estimation, and the services it renders to the State and to the individual will bring a full return of appreciation and remuneration."

On the conclusion of the address, Major-General THOMSON, C.B., in moving a vote of thanks to Mr Hunting, said that he had listened with the very greatest pleasure to Mr Hunting's words. He felt sure that all would agree with him that those words had been instructive as well as interesting. The lecturer had in the most lucid way taken them back to the early history of the profession, to that early time when veterinary science could not be said to exist in the country as a profession, till a Frenchman arrived to teach them a few fundamental lessons. Mr Hunting had shown them how, from that time onward, the profession had, by short strides and by long strides, continued to improve slowly but surely up to present times. He had shown also the results achieved by the efforts of veterinary science in the suppression of contagious animal diseases; he had shown what difficulties the profession had had to meet in conducting that suppression, what prejudice and ill-will had for a long time impeded them, till there were found sensible men to see the necessities of the case with a clear eye. From that time onward, as Mr Hunting had told them, the profession had held a position incomparably above its former one, a position which had continued, and would continue, to increase in importance. He had suggested what measures the profession should take for its continued improvement, and what duties it might expect to assume in the future. As one measure to secure improvement he had suggested that the Government, through the Board of Agriculture or some other Board, might be asked to support them by endowing a bacteriological laboratory and other things necessary for their work; that was one thing which the profession, as a poor one, were bound to obtain in the end. He thought it unjust, nay, absolutely scandalous, that a wealthy Government and a wealthy nation such as their own, should look to private individuals to initiate what was really a very great national necessity. That, indeed, was the real state of affairs, as they would see if they considered the case, for instance, of the College in which they were then present. Did it obtain anything from the Government? Absolutely nothing, and it never had received anything. No veterinary college had the support of the Government in any way whatsoever. On these grounds he entirely endorsed Mr Hunting's conception of the duty of the Government to help in that matter. He had listened to the address with great pleasure, and had even greater pleasure in proposing a vote of thanks to Mr Hunting.

The CHAIRMAN then put the vote of thanks to the assembly, and it was carried with hearty applause.

Mr HUNTING, in acknowledging the compliment, said that he felt deeply indebted to the assembly for that mark of their approbation. Even without it, he would have felt but too proud and too pleased to have had the opportunity of addressing one of the largest audiences—certainly the youngest—that he had ever faced.

At this stage of the proceedings the prize list for the previous session was read by the Principal (*see* p. 284).

Sir GEORGE BROWN, C.B., recalled the old saying that one should never abuse an enemy behind his back nor flatter a friend before his face. His object in rising was to propose a motion which they would all receive with heartiness—a vote of thanks to Sir Thomas Elliott for his conduct in the chair that day. Bearing in mind the second part of the injunction which he had just uttered, he felt unable to express much of what his own personal feelings would lead him to say; but he might at least remind them that Sir Thomas Elliott had proved himself a great friend to members of their profession, and that he had at that time a much larger veterinary staff than had ever before existed under the direction of a government department in the history of England. Further, he represented agriculture, and some of his own old pupils then present would recollect that he (the speaker) had always harped on that one string—the vast importance of a closer association between agriculture and the members of the veterinary profession. That point had been illustrated by Mr Hunting, who had impressed upon them very strongly the value of hygiene and the great importance of preventive medicine. He had also told them something of the work which had been done by the old veterinary department, now associated with the agricultural department; and he might mention as a salient point in the whole of the proceedings that their great success had been due to a dogged determination not to relax restrictions merely because a particular disease was declining. Sir Thomas Elliott would recollect well how deputations had come to the Board of Agriculture and urged very painfully indeed that "now that the disease had nearly ceased, surely he might remove some of those serious restrictions." The answer had been that "then was the time that they could definitely rid themselves of the disease. They had got the enemy down, and proposed to tie his arms and legs and jump on him." There was no such thing as "fair fighting" when dealing with disease, and he would strongly urge them to rely a great deal upon what Mr Hunting had said with regard to the importance of preventing disease. He remembered how the late John Ruskin, in one of his early essays, in describing the duties of the members of the commonwealth, said that it was the duty of the merchants to feed them and of the physicians to keep them in health—not to cure them of disease, but to keep them free of it. Mr Hunting had spoken of the achievement of a former Minister of Agriculture—the Hon. Walter Long—in stamping out rabies. He (the speaker) knew that Mr Long's life had been made as unhappy as the life of an official could well be made by urgent solicitations, and sometimes stern demands, to relieve the public from that muzzling order. But he had calmly looked on while they were abusing him, and sat tight till all the risks had passed. It was on the point of small risk that so much mischief had been done. People often said, "there is so little in the matter that it is not worth taking trouble about it; let them be set free." Even a great pathologist had spoken of the slight danger of communication of tuberculous disease from a bovine source. He was inclined to agree with him, but now was the time, the risk being slight, to get rid of the disease altogether. He never heard the argument to abandon effort to get rid of a slight risk, without thinking of the story of the nervous gentleman who lived in a mountainous country. He was always afraid that his coachman would drive him over the edge of a precipice, so he determined to try a new man, and advertised for a very steady and careful coachman. One man said he would go within two inches of the edge, and another wanted a foot; so they differed very largely. Presently there came along an Irishman, and with that "cuteness" which was common to the people of that country, he was equal to the occasion. He was asked how near he could go to the edge of the precipice without running any risk of falling over; he seemed rather surprised at the question, and after a few moments' hesitation replied, "Sure, I'd keep as far from it as ever I could."

Sir Nigel Kingscote, K.C.B., rose to second the vote of thanks to Sir Thomas Elliott. He was not going to detain them a minute, but as a Governor of some standing in the college it was a matter of great pleasure to him to be there, and to have had the advantage of listening to such an interesting and instructive address as that delivered by Mr Hunting. As they had heard, the Governors of the college had had by no means an easy task in conducting its operations, and their task was rendered even more difficult at that time. There was great need, as Mr Hunting had suggested, for an appeal to the Board of Agriculture or some other body to help them. It had been a great source of satisfaction to himself, and, he felt sure, to all the Governors, that Sir Thomas Elliott had taken the chair that day. He felt certain that his (Sir Thomas Elliott's) sympathies were entirely with them, that he fully recognised the value of the veterinary profession, and that he would do all in his power to keep that college in the state of efficiency in which he believed it to be at that moment. To keep it in that state was their pride. He begged to thank Sir Thomas Elliott for his presence there that day.

Sir THOMAS ELLIOTT, in rising to acknowledge the compliment, assured them that he needed no vote of thanks for anything he had done that day. It had been a great pleasure to him to be present to hear Mr Hunting, whose address had been extremely engrossing, and full of matter of the greatest interest to him as secretary of the Board of Agriculture. Probably if Mr Hunting and himself were to discuss every portion of his address it would prove a very pleasant work, certainly they would become very hungry and thirsty in the course of the discussion. But in particular the portion of his address with which he wished to associate himself was that in which Mr Hunting had referred to the great debt which agriculturists had been under in the past, were in the present, and would be, he hoped he might say, in the future, to the veterinary profession. No one knew better than himself, none had had a better opportunity than himself, of forming some idea of what the condition of agriculture would have been, had it not been for the labours of the veterinary profession. Veterinary surgeons, with their science and their research, had been able to devise means by which disease could be stamped out. At a time like the present, when the question of profit and loss in agricultural matters was a matter of very small balancing on either side, he thought it needed no very great imagination to see that if farmers and stock-holders were subject to losses by disease in the present such as they had suffered in the past, the position of farming in this country would be almost impossible. It was a great pleasure to him that the vote of thanks on that occasion had been proposed by his old friend and colleague Sir George Brown. Mr Hunting had referred to the services rendered by Sir George to the cause of veterinary science in the past, and especially in his vigorous efforts in the direction of eradicating swine fever. He had been the pioneer in that work, and everybody knew how dearly his memory was cherished at the Board of Agriculture. He also saw another friend of his there, some of his present colleagues. and might he also say, some of those who he hoped would be his colleagues in the future. It was perfectly true that as long as the work of the Board of Agriculture continued, as long as there was a large, skilled, and he hoped, well paid staff, they would have to recruit that staff from time to time, and he could only hope that some of those present would look forward in that direction, and by their special work in that college, qualify themselves to join those at the Board of Agriculture. But whether they did so or not, at any rate they had the satisfaction of knowing that they were to become members of an honourable and beneficent profession, which they must do their best to advance.

The proceedings then terminated.

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